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A CASE OF NEUROFIBROMATOSIS WITH BUPHTHALMUS ALFRED WIENER

(Complete contents inside)

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ARCHIVES OF OPHTHALMOLOGY.

OBSCURE AFFECTION OF THE CONJUNCTIVA RESEMBLING CASES OF PARINAUD'S SYNDROME.

By Major R. E. WRIGHT, I. M. S.

Superintendent, Government Ophthalmic Hospital, Madras.

THE patient was a Hindu fireman aged 32. Early in March, 1924, while the train was in motion a spark flew into his left eye. Six days later the eye became red and was attended to by the railway doctor. In another week the redness became more intense and a painless swelling appeared in front of the left ear. He was sent to a hospital where he was treated for 12 days and was transferred here on April 4th, 1924, practically a month after the condition started. During all this time he was not aware of any constitutional or febrile disturbance. The right eye was red and swollen and the upper lid drooped. The conjunctiva at the angles was chemosed, and a thin creamy purulent discharge was present. At first sight, the eye looked like a bad case of ophthalmia due to a mixed infection, following gonorrhœal conjunctivitis. On everting the lid large cocks-comb-like granulations bulged forward. These covered the whole of the tarsal conjunctiva and extended to the fornix and to a lesser extent to the bulbar conjunctiva. Here they were smaller and paler. The lid margin was rounded, and in places granulations extended on to it. The limbus was swollen all round and one or two granulations were present there also. There were paler areas here and there about the size of millet seeds, not purulent, but suggestive of avascular cell aggregations which showed up a lighter pinkish yellow in the surrounding deep red. The eye was suggestive of tuberculous conjunctivitis, but there was no ulcer or sinus to be seen, and no breach of surface. Smears

from the conjunctival discharge were found negative for microörganisms. The von Pirquet test was positive, but there were no clinical evidences of tuberculosis. The Wassermann test was positive strong and there was a history of syphilis, with clinical evidence. The patient was put on cod liver oil and iron, and a course of novarsenobillon was commenced. The conjunctiva was treated by means of frequent irrigations, dry heat, and silver nitrate applications (1%). Subsequently, bacillary emulsion was given, starting with a dose of $\frac{1}{8000000}$ *mg*, and mercury was substituted for the novarsinobillon combined with large doses of potassium iodide. The patient did not improve under treatment although everything possible was done for him. The granulomatous tissue spread over the bulbar conjunctiva and on to the cornea. By the 11th May the granulations had invaded the substantia propria until the latter became so thinned out that a keratocele developed. This was practically a month after his admission. During this period attempts were made to obtain cultures from the conjunctival sac, but nothing could be grown. Guinea pigs were inoculated with negative results. The patient was taken to the King Institute, Guindy, and cultures made by the staff with negative results. On 26–5–24 X-ray exposures were commenced, but in spite of treatment the granulations extended all over the cornea and the keratocele increased in size. The patient would not remain and took his discharge and presumably the eye was lost. Portions of the granulomatous tissue were obtained from the lid on two occasions and different portions fixed in Schaudin's fluid, Zenker's fluid, and formol. Some of these pieces were subjected to serial sectioning and staining. In one portion of tissue only could anything suggestive be seen. In sections through this piece of tissue fairly numerous giant cells were to be observed as well as endothelioid cells, plasma cells, and fibroblasts. It was in other words a chronic granulomatous type of tissue, but no irritant could be discovered to account for this reaction. As was expected from the animal experiments, tubercle bacilli were not to be seen. Special staining was adopted for other types of higher organisms without result.

Professor Verhoeff was communicated with in this connection and very kindly furnished stained slides of the leptothrix conjunctivæ which he has described. With reference to the case under report he remarked that in a specimen sent to him, the appearances did not at all resemble those met

with in the type of case which he has described as due to the leptothrix conjunctivæ. It seemed to me possible that the condition was one of chronic granulomatous change due to one of the higher bacteria, but this could not be proved. This suggestion was emphasized, as during the latter part of the investigation of the case, a chronic granulomatous condition of the orbit (a note on which will subsequently be published in detail) was proved to be due to an aspergillus.

The present case is reported as possibly falling within the class of Parinaud's syndrome. It does not resemble Pascheff's cases, and differs from Verhoeff's conjunctivitis and many other recorded cases in that it progressed to the destruction of the cornea and eventually no doubt to a total loss of the eye. The main features may here be summarized. There was a definite history of trauma, no evidence of animal contact. Constitutional or febrile disturbances were uncertain; the glandular involvement was slight. Only the preauricular gland was swollen and it subsided after three months. The local lesion showed large coarse polypoid granulations with grayish yellow areas, without ulceration. The granulations eventually invaded the cornea and destroyed it. Animal experiments and cultural experiments were negative. Anti-syphilitic treatment with novarsenobillon, mercury, and potassium iodide, X-ray treatment, and anti-tuberculous treatment all proved of no avail. The only case met with here of recent years closely resembling the above was one of conjunctival tuberculosis verified by microscopic examination.

REPORT OF A CASE OF ORBITAL NEURINOMA.[1]

By Dr. MARTIN COHEN, New York.

(With four illustrations on Text-Plate XVI.)

THE following case is presented because of the few instances of this type of tumor reported in the literature.

An eleven-year-old boy, W. F., was first examined by me on January 8, 1924. He has always been well, and his parents and three sisters who are living are all in excellent health. The general physical examination was normal. No nodes were felt in any part of his body. The X-ray, tuberculin, and Wassermann tests were negative. No important diseases of childhood have occurred. The boy is very intelligent.

When the boy was nine years old, his father first noticed that there was slight protrusion of the right eyeball which gradually became more prominent. When first examined this eye was markedly proptosed with an exophthalmos of 10*mm;* the exophthalmometer reading was 23 in the right eye and 13 in the left eye. Externally the right eye except for the exophthalmos and a slight dilatation of the palpebral fissure was normal; the pupillary reaction, the field of vision and the intraocular tension were normal. The vision was $\frac{20}{30}$, and slightly myopic. This eyeball protruded straight forward with very slight motility in all directions. Palpation of the eyeball met with a soft resistance; the orbital rim was free from any involvement. The fundus examination showed a choked disk of seven dioptries with engorgement and tortuosity of the retinal veins. The arteries were normal. No hemorrhages nor exudates were present. This choked disk was a late manifestation and was probably due to the pressure of the growth on the intervaginal space. The left eye was normal in every respect.

[1] Read before the Section on Ophthalmology of the New York Academy of Medicine, May 19, 1924.

ILLUSTRATING DR. MARTIN COHEN'S ARTICLE, "REPORT OF A CASE OF ORBITAL NEURINOMA."

FIG. 1. Gross tumor, 35 x 22 mm. Cut surface showing hemorrhage under the capsule and scattered throughout. A. Pedicle 3 mm. in diameter.

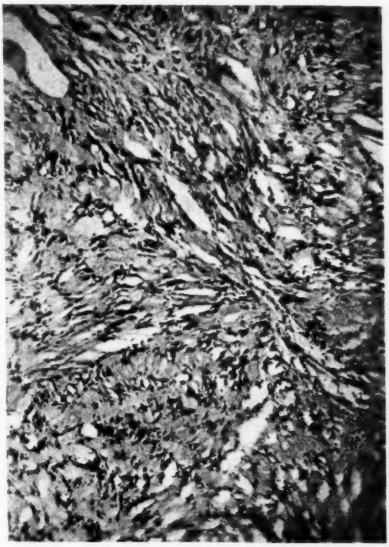

FIG. 2. Low power photomicrograph showing elongated spindle cells arranged in bundles twisting about in various directions. The dilated lymph spaces are numerous. (See A).

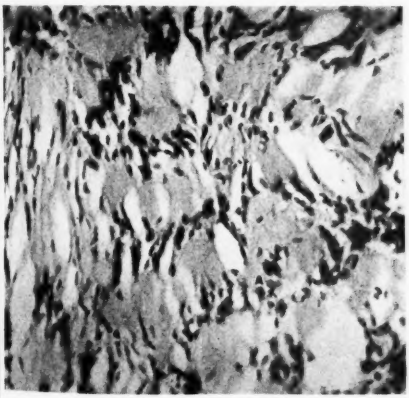

FIG. 3. High-power photomicrograph of (A) in Fig. 2 showing the sheath of Swann cell nuclei arranged in columns like soldiers "on parade," also called a palisade arrangement (A).

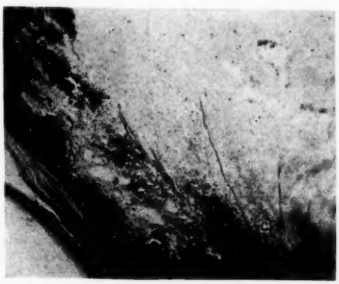

FIG. 4. Low-power photomicrograph showing capsule (A) hemorrhages beneath the capsule (B) and medullated nerve fibres at the periphery of the tumor (C). (Pal Weigert without counter stain).

The diagnosis made was: primary retro-orbital benign growth in the right orbit, located within the cone of muscles. A tumor of the optic nerve was not considered, as the vision remained normal for two years, although the position of the eyeball was suggestive for an optic nerve tumor. Operation for the removal of the growth was advised on account of the rapidly increasing marked exophthalmos. The Kronlein operation was performed on January 13. After the usual incision the periorbital tissue was incised; it exposed a mass which could be readily felt with the finger. It was soft in consistency, pear-shaped, movable, apparently encapsulated, and about the size of a walnut. This mass extended from a level below the eyeball along the outer side of the optic nerve to the apex of the orbit around the annulus of Zinn.

With blunt dissection, the mass was separated from the surrounding tissue; it appeared to be attached to the apex of the orbit close to the optic nerve sheath. This attachment was severed with the scissors and the mass placed in formalin solution for future miscroscopical study. Following the operation, the lids were sutured together and the eye bandaged. The bandage was removed for the first time four days after the operation and the eye inspected. The vision equalled motion of fingers now at two feet; and after one week there was no light perception. The eye was markedly converged, due to a traumatic paralysis of the external rectus muscle. There was anæsthesia of the ocular conjunctiva and cornea due to partial involvement of the fifth nerve; there also appeared a linear infiltration in the lower quadrant of the cornea. There was no direct nor indirect pupillary light reaction, on account of the secondary optic nerve atrophy. The motility of the eyeball was regained except in external rotation, due to the traumatic paralysis.

The fundus examination showed the disk to be diffusely grayish in color; its margin was slightly blurred, with nonappearance of the lamina cribrosa. There was a slight increased pigmentation about the margin of the disk, indicating secondary optic nerve atrophy; otherwise, the fundus appeared normal. The secondary optic nerve atrophy was probably due to a retrogression of the choked disk. As the eye was markedly converged, I performed a tenotomy of the internal rectus, and advanced the external rectus muscle with satisfactory results.

Microscopical Report.—Gross. Specimen is a smooth, oval-shaped body 35*mm* in length and 22*mm* in diameter. It is fairly soft and elastic. It is well encapsulated except at one end, where there is a small denuded area 3 x 4*mm*,

apparently the pedicle. The capsule is dark red with purple and gray areas. On section there are extensive hemorrhages beneath the capsule. The tumor proper is opaque, pale gray with red hemorrhagic areas and yellow markings.

Microscopic.—The tumor is covered with a capsule 1*mm* thick, composed of fibrous tissue lamellæ. A few fibrous tabs are attached to the outer surface. Fairly large blood vessels course through its substance. Rather extensive hemorrhages are seen beneath the capsule and suffuse the tumor proper to some extent. This extravasated blood obscures the tissue structures and the relation of the capsule to the tumor proper, but fibers from the capsule appear to extend into the substance of the tumor.

The tumor is fairly uniform in structure throughout. It is composed of elongated spindle cells that form bundles twisting about each other in various directions. The nuclei are frequently in parallel rows, "palisade formations" forming compact groups separated by considerable areas formed entirely of fibrils. The structure is more solid and the nuclei more numerous near the pedicle mentioned in the gross.

The nuclei vary very greatly in size and shape; the predominating form is large oval or spindle shape. They vary in staining property; many are pale and many are deep in color. Between the bundles and within them there are numerous dilated lymph spaces, giving the tumor its somewhat soft consistency. Blood vessels are fairly numerous. They are thin-walled and dilated with occasional extravasations of blood about them.

With special stains medullating nerve fibers are found beneath the capsule, a few passing into the tumor where they end abruptly before reaching any depth. Special stain also shows fat droplets in many cells. These cells are irregularly distributed and bear a relation to the hemorrhagic areas in part, and in part are found in other localities, suggesting phagocytosis of degenerating nerves. It is collections of these fat-staining cells that give the yellow color to areas in the gross.

The Van Gieson and Weigert-Pal stains point to a nerve origin with poorly-differentiated nerve cells as a chief component, mostly sheath of Schwann cells. These tumors are usually attributed to an embryonic defect in development. This tumor is not unlike many of the reported tumors of the optic nerve and moreover has a relation to the neuro-fibromata of the older pathologists. These nerve tumors are rare in any

locality and there has been considerable work done to clear up the classification of the various forms which seem to belong to closely related groups.

In 1910 Verocay made an exhaustive study of neurofibromata in an effort to determine their histogenesis. One of the results of his study was a re-grouping of nerve tumors that had previously been named "neurofibroma." Among them was a group called by him "neurinoma." This group had special features to distinguish it,—features that are present in the tumor under consideration.

Since Verocay's publication there has been general acceptance of his work, and now only tumors formed of proliferating perineurium and endoneurium bear the name "neurofibroma."

The special histological features in our case are:

The palisade arrangement of the nuclei of the spindle cells, —that is, the sheath of Schwann cells,—in many areas and the tinctorial reaction of the Van Gieson stain. Tumors with these details cannot be called simply neurofibromata.

Diagnosis.—Neurinoma "Verocay" (a tumor classed as neurofibroma by the older school of pathologists).

This tumor from the pathological point of view is a nerve tumor, but not of the optic nerve for the following reasons:

First: On account of the preservation of excellent vision for several years while the exophthalmos existed. Second: The presence of numerous sheath of Schwann cells which do not exist in the optic nerve. Third: The presence of the tumor mass, being palpable and movable but not attached, along side of the entire course of the optic nerve during the operation.

From these facts, and its situation in the orbit, it necessarily must be a nerve tumor of one of the branches of one of the other cranial nerves, either the third, fourth, sixth, or ophthalmic branch of the fifth nerve.

It is naturally impossible to state positively from which nerve the tumor originated; but from the symptoms of the ocular nerve condition, it gives me the impression that the tumor originated from a branch of the posterior ciliary nerve.

I wish to express my thanks and appreciation to Dr. Louise A. Meeker for the pathological report in this case.

ACUTE DACRYOADENITIS CAUSED BY FOCAL INFECTION.[1]

By Dr. MATTHIAS LANCKTON FOSTER, New Rochelle, N. Y.

THE paucity of literature on acute dacryoadenitis in its early stage, the opportunity presented to observe certain very early symptoms, and the peculiar etiology, which seems to me to be demonstrated, impel me to place the following case on record.

A gentleman 27 years old was wakened one morning about 3.30 by a terrific pain in his left eye. At 9 o'clock both lids of his left eye were slightly reddened and swollen. The bulbar conjunctiva presented two vascular red bands starting near the cornea and growing more and more deeply red toward the fornix. One of these led outward and upward beneath the lid toward the accessory lacrimal gland, the other more upward and outward toward the site of the lacrimal gland proper. On everting the upper lid an irregular, very tender mass protruded, which exactly resembled a swollen accessory lacrimal gland covered by conjunctiva. The angry redness of the upper band of engorged blood vessels suggested the presence of an irritating foreign body lodged in the upper fornix, but a careful examination revealed no foreign body. Touching the fornix at the place of greatest redness with a swab of wet cotton disclosed an area of great tenderness. Palpation with the finger beneath the brow revealed an indistinct sense of resistance and caused much pain. These findings seemed to justify the diagnosis of acute dacryoadenitis, although there was no apparent exophthalmos or deviation of the eyeball in any other direction, no diplopia, and no chemosis at that time. The upper lid could be everted and then lifted to expose the fornix nearly as well as with an

[1] Read before the American Ophthalmological Society, May 4, 1925.

uninflamed eye. The etiology was obscure. The man was apparently in perfect health, had not recently had, or been exposed to, mumps, gonorrhœa, or any other contagious disease, had not been unduly exposed to cold, and had received no trauma. The treatment prescribed was an active catharsis and hot compresses locally, changed every minute for ten minutes every two hours.

On the following day the lids were less swollen, the pain less severe. The left pupil was smaller than the right and the patient stated that the sight of the eye was blurred. An apparent myopia of 1.25 D was present, but was quickly dispelled by homatropine, which brought the vision up to $\frac{20}{20}$ without a glass in a short time. Irritation of the ciliary body was thus added to the symptoms.

During the next few days the pain and the swelling of the accessory lacrimal gland slowly but steadily subsided. Persistent investigation had revealed the presence of pivot teeth and bridge work, but the patient was averse to considering his teeth at all while matters seemed to be doing well with his eye.

Six days after the first attack an exacerbation of pain occurred during the night. In the morning there was a slight exophthalmos, the conjunctiva was chemotic, there was no tenderness over the region of the lacrimal gland and no gland could be felt by palpation, but a tender point had appeared in the orbit directly below the eyeball. A roentgenogram now made of his teeth revealed abscesses at the roots of the two left upper incisors, which were capped with pivots. These were extracted. The improvement was so rapid that practically no signs of the ocular trouble persisted until the next day. The patient claimed that he could feel the pain subside from the moment the teeth were out. There has been no recurrence. The entire inflammatory process in the orbit seems to have been dependent on the focal infection at the roots of the teeth.

A CASE OF "KERATITIS PUNCTATA SUPERFICIALIS."

By Dr. A. W. STIRLING, Atlanta, Ga.

THE following case, which at the time of her first visit I noted as *keratitis punctata superficialis*, may perhaps with advantage be added to those reported by Dr. L. R. Gifford in the Archives for May under the title "Epithelial Dystrophy and Recurrent Erosion of the Cornea."

A married, childless lady, age 26 years, consulted me in July, 1923, on account of pain and a constant sensation of foreign bodies in the eyes. There had been previous attacks, which had apparently partially or completely subsided, and she had been under the continual care of a first class ophthalmic surgeon for six months. He had prescribed collyria, salves, Fowler's Solution internally, and she wore glasses. Some unhealthy teeth and her tonsils had been removed. Her health was good, but she "was nervous."

Both corneæ were covered by superficial dots, in the manner shown in Dr. Gifford's illustration of the right eye in Text-Plate VIII, and the upper lids were red and swollen.

I treated those eyes and lids with the utmost care for three months, and with everything I could think of; I had her examined thoroughly from top to toe (including her nose, of course), and in October I could not claim to have done her one iota of good.

In the meantime I had been corresponding about her stubborn condition with Mr. Treacher Collins and Dr. J. M. Wheeler, with the result that I first applied pure carbolic acid to the spots, without benefit, and later, on October 18th, scraped the left cornea clean of epithelium and painted it with tincture of iodine. I had warned her to prepare for trouble, and in spite of an ointment containing atropine,

cocaine, and acid boric, she was not disappointed. When she returned in five days she had forgiven me especially as the raised spots were gone leaving only slight opacities in their place. On the 30th the left eye seemed to be well; there was no discomfort, and the opacities had gone. At her request I used the like drastic treatment in the other eye.

I did not see her again until November 27th. It appears that the right eye had pained her much more than the left, and also that a few days after the little operation, she had been attacked by grippe which kept her a week or so in bed.

The right eye was quite free of spots. On the left were two of minute size. Her eyes felt "quite well."

I prescribed glasses:

R. + 1.00 + 1.00 cyl. axis 105 degrees, V = $\frac{6}{6}$ −.
L. + 0.25 + 1.00 cyl. axis 180 degrees, V = $\frac{6}{6}$ −.

On June 23, 1924, the patient returned very despondent. The irritation was back, and the centers of the corneæ were again covered by spots. She was leaving the city, and was in two minds when I suggested repeating the performance on her return. I have since seen her husband several times, and understand she is still suffering, but apparently not enough to justify her in her own estimation in having the corneæ scraped.

Later—It has been with some difficulty that I have at last located this patient. Her husband was one of the flock of migrants to Florida.

She writes me as follows:

"Your letter of July the tenth received, and am sorry to state that I am still having trouble with my eyes, sometimes, I think, more than ever. I would not advise the scraping of eyes and burning out with iodine or carbolic acid."

One of my chief reasons for bringing this case before the profession is the hope that some one may be able to suggest a course of treatment holding out a prospect of permanent relief.

NOTES ON THE GENESIS OF MYOPIA.

By Prof. G. LEVINSOHN, Berlin.

IN an essay which I recently published in the *Klin. Monatsblätter für Augenheilk.*,[1] I referred to the imperfections in the work of Steiger[2] and showed how improbable his conclusion is, that myopia is a refractive anomaly due entirely to heredity. It can be proved that outside factors play a very important part, and that these factors must be sought in the bending of the body and the head. But as even in the latest publications about this theme the importance of my opinion is absolutely misconstrued, permit me here to sum up the material on which my opinion is based and which in my estimation furnishes incontestable proof of the correctness of my idea.

The fact that in cases of high myopia an elongation of the eyeball takes place, is now admitted even by those authors who from Steiger's point of view trace the cause of myopia to heredity only, e.g., by Vogt[3] and Siegrist.[4] Without ado it is evident that there is a contradiction between these authors and Steiger, for Steiger claims special credit for having proved that the axis of the eye in cases of myopia can never be pathologically lengthened, but that the eye, without external cause, develops those curious forms which we see in cases of myopia.

Since the striking changes in cases of high myopia lead us to

[1] G. Levinsohn, "Zur Frage der Myopiegenese," *K. M. f. A.*

[2] Steiger, *Die Entestehung d. spharischen Refraktionen des Auges*, Berlin, 1913.

[3] Vogt, "Zur Genese der Spharischen Refraktion," *Berichtd. D. O. G.*, Heidelberg, 24.

[4] Siegrist, *Refraktion u. Acc. d. Auges*, Berlin, 1925.

consider outside factors as causes for the extension of the eyeball, this supposition is also valid for cases of myopia of less severity. One must without further ado concur in Steiger's opinion, that under such a supposition even in many cases of normal-sighted and long-sighted persons a pathological increase of the axis can be supposed. This supposition, which, according to Steiger, seems to be impossible, is indeed proved by facts. It is not possible to draw a sharp line between the various degrees of refraction, in particular not between the high, less severe and low degrees of myopia. One refraction passes directly on to the other, and the clinical and anatomical changes which we see in particular in cases of high myopia, less in cases of low myopia and very seldom only in cases of normal-sightedness and long-sightedness can either resemble each other completely or—and so it is in general—distinguish themselves from one another only gradually. Above all, these changes are clinically expressed by the cone or the connective-tissue ring. Nearly always both forms are identical and differ only with respect to the grade of extension. By comparing the background of the eyes of newborn babes, children and adults I could show[1] that not only the cone but also the connective-tissue ring, which generally will be looked upon as a variety of the entrance place of the optic nerve, in most of the cases are not hereditary, but acquired and must be looked upon as a result of an extension of the eyeball.

That this extension cannot be caused by an increase of pressure can be shown by the fact that no such pressure increase exists during convergence, and that the changes which are characteristic of myopia altogether differ from glaucomatous changes. Hess and Heine have proved that increase in tension is not present during accommodation, while I furnished[2] proof that the activity of the various eye muscles during accommodation likewise leaves the pressure unchanged or increases it so slightly that this increase of pressure cannot be supposed to change the coat (wall) of the eyeball. Wessely corroborated the extremely small pressure

[1] G. Levinsohn, "Zur Myopiegenese," *K. M. f. A.*, vol. 66, p. 84, 1921.
[2] G. Levinsohn, *Graefe's Archiv*, vol. 76, p. 129.

increase on the living eye when the eye is converging. If therefore Siegrist says in his newest publication,[1] "It seems to me that it can scarcely be doubted that such (myopic) changes may result not from the accommodating muscle but from a chronic compression and pulling of exterior eye muscles," he makes the same fundamental error that all present supporters of the muscle-theories have done. For the rest, a very simple example will show the impossibility of this idea. Let us fill a rubber ball, a circumscribed place of the coat of which is about half as thick as the rest of it, with water and then increase the pressure inside the ball by pressing on it, it is quite evident that the thinned spot of the coat will be bent outwards first. Just in the same way every increase of pressure in the eye must, and will always affect the lamina cribrosa first, and the remaining part of the external coat only if this is less resistant (infantile glaucoma). The same is true concerning the theory of Stilling, which for this reason must be looked upon as wrong, as it is based chiefly on the factor of growth; the growth of the eye however playing a very unimportant part after the fourth year of life.

In the face of these thoroughly insufficient myopia theories the author of this treatise has shown that it is not the near-use of the eyes per se, but the bending of the head and the body going along with the accommodation that must be responsible for the extension of the eyeball. This conception has been confirmed by theoretical reasons, and reasons resulting from actual practice.

Theoretical Reasons.

(1) When the body and the head are bent the eyeball will be pushed downwards into the orbit on account of greater congestion, and because of its own gravity. As the eyeball essentially represents an extensible ball filled with water, an extension of the ball must be the result of the above-named movement.

(2) The pulling of the external coat will naturally and preferably work on the place where the eye is fastened to the optic nerve, namely the place of the entrance of the same

[1] *L. c.*

and the surrounding area. As the optic nerve runs from the nasal to the temporal side, the gravity of the eyeball mobilizes forces which: (1) Cause an extension of the eye in a sagittal direction, (2) affect the temporal margin of the optic nerve very much, and (3) tend to displace the nerve entrance to the nasal side. In this way the characteristic anatomic changes in cases of myopia (namely, lengthening of the sagittal axis, thinning of the sclera on the posterior pole, retraction of the lamina elastica with the pigment-epithelium, atrophy of the choroid, bending of the optic-nerve fibers on the temporal side, supertraction of the choroid and the retina on the nasal side) are made plausible.

(3) The structures which tend to hold the eye in place when the body and the head are bent are not able to prevent the eyeball from falling forwards. These structures are the lids, the connective-tissue which fastens the eye to the bony surroundings, the numerous nerves and vessels entering the eye, the muscle funnel and the optic nerve. The lids are very extensible and by opening the palpebral fissure they easily give way to the pressing eyeball. The conjunctiva, the connective tissue membranes and the vessels and nerves in a smaller degree are likewise extensible organs. The extensibility of the muscles is, as shown by the experiments of Landois, by far too great to allow them to work effectively against the falling forward of the eye. And what resistance is offered by the optic nerve? L. Weiss has shown that the optic nerve is not bent during accommodation, that is during convergence, but appears more or less stretched, so that when the head and the body are bent the weight of the eye can act on the nerve. Under such circumstances one must expect that the force of gravity brought about by the bending of the head, will have a pulling and extending effect on the living optic nerve itself, even if the nerve is bent.

A hitherto puzzling fact, namely the fact that in certain categories of working men such as jewelers, watchmakers and embroiderers in spite of intense near-work the percentage of myopic cases is relatively low, coincides with my myopia theory. It can be proved that the eyes of these workers were near their work, but their heads and bodies were bent relatively little.

PRACTICAL REASONS.

(1) Experimentally I have furnished proof that when the body and the head are bent the eye will fall forward more or less. These investigations are identical with those of Birch-Hirschfeld, who determined the same condition by means of photographs, and with those of Dinger whose methods of investigation are similar to mine. Furthermore it can be proved that the power which, when the head and body are bent, pushes the eye forward, consists of two components, the increased congestion of the orbit and the gravity of the eyeball.

(2) Of greatest importance however in determining the etiology of myopia is the experimental proof that, in accordance with my theory, by bringing the sagittal axis into a vertical position, an artificial myopia can be produced in animals, and a myopia once existing can be increased in this manner. First these experiments were made with young dogs, cats and rabbits. Although these experiments gave positive results, they were later on renounced, as the experimental technique with these animals made cruel methods necessary. Then the experiments were repeated with monkeys and with other methods. With regard to the fact that these last experiments have been considered as extremely cruel, it may be said here once more, that these were extraordinarily harmless experiments, the admissibility of which should be acknowledged even by those who object to all animal experiments as cruel. My experiments with monkeys have furnished full proof that the horizontal position of the eye is sufficient to produce an axial myopia in animals in a relatively short time (several months), and this myopia will increase the longer the experiment lasts,—that furthermore along with the increase of the myopia ophthalmoscopic changes will take place such as are seen in cases of human myopia, and finally that the anatomic substratum of this experimentally produced myopia is identical with the anatomic changes of the human higher myopia. Behr's[1] experiments with monkeys can be valued as controlling experi-

[1] Behr, *K. M. f. A.*, vol. 72, 1919.

ments only, as they were stopped after two weeks. In accordance with my experiments no extension of the eyeball can be expected with certainty to develop within such a short time. On the other hand Behr's negative result[1] on an especially disposed material, watched during several years, is sufficient to show very clearly the effect on the eyes of a horizontal position.

The proof that in cases of myopia the extension of the eye is caused chiefly by the bending of the body and the head is so clear that it is high time to draw the obviously important hygienic conclusions from this fact.

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[1] G. Levinsohn, *K. M. f. A.*, vol. 72, p. 744, vol. 1919.

THE NON-SURGICAL TREATMENT OF HETEROPHORIA IN AMETROPIA[1]

By Dr. HERBERT WRIGHT WOOTTON,

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THE varieties of heterophoria which will be considered in this paper are esophoria, exophoria, and hyperphoria, and the first two will be treated as latent anomalies of convergence and divergence, not as distinct pathological entities. This is the viewpoint from which they were regarded first, by Duane, and this method of regarding them marked the beginning of treatment leading to practical and definite results. It is very true that the first latent anomaly described was called by von Graefe latent divergence and not exophoria, but this fact was for the most part lost sight of, in this country at least, and the nomenclature of Stevens was later adopted. Stevens regarded esophoria and exophoria as distinct diseases of the ocular muscular system, due at times to refractive errors but even more frequently to over-action of individual muscles. He, therefore, directed his treatment towards esophoria and exophoria *per se*. Duane attempted to prove and, in the writer's opinion, did prove conclusively that esophoria and exophoria were not diseases, but merely symptoms, and that they depended not upon excessive action of individual muscles but upon excessive or insufficient action of convergence or divergence. Thus, according to Duane, the symptom esophoria might be due either to an excess of converging power or an insufficiency of diverging power, and obviously the treatment of two such diametrically opposed conditions,

[1] Read before the Ophthalmological Section, New York Academy of Medicine, April 20, 1925.

even though they caused the same symptom, ought not to be the same. In similar manner, exophoria might be due either to a divergence excess or to a convergence insufficiency, and here again the treatment would differ in the two cases. It seems to the writer that the validity of Duane's position has been firmly established and that we would do well to call these horizontal latent anomalies by terms expressing their dynamic status rather than their static, to speak of a convergence excess or divergence insufficiency rather than of an esophoria and of a convergence insufficiency or a divergence excess rather than of an exophoria. The attitude of the writer, in regard to these conditions having thus been stated, we are now ready to consider their non-surgical treatment.

I. Convergence Excess. This condition, one form of esophoria, is an almost constant accompaniment of an uncorrected hypermetropia. It is caused by the hypermetropia and does not give trouble after the hypermetropia has been properly corrected. Its non-surgical treatment consists in the prescription of convex lenses.

II. Convergence Insufficiency, one of the forms of exophoria. This condition may be accompanied by myopia and the correction of the myopia may cause its disappearance. Cure doesn't always follow the prescription of concave lenses, however, and the condition may be associated with hypermetropia, a state of affairs in which the prescription of convex lenses will tend rather to aggravate the symptoms due to the muscular error. The non-surgical treatment of these cases consists in attempting to increase the converging power by means of exercises with prisms, base out or, in those cases associated with hypermetropia in which the glasses are needed only for near work, we may add prisms of low degree, base in. Prismatic exercises frequently succeed at least temporarily and, of course, they can be repeated from time to time as the symptoms recur. The addition of prisms to convex lenses, for near work only, while perhaps a subterfuge, nevertheless frequently makes the patient comfortable and enables him to avoid advancement of an internus. Its ultimate effect upon the muscular error would be theoretically to make it worse.

III. Divergence Excess. This condition, one form of exophoria, rarely causes symptoms until it begins to pass over

into divergent squint when the transient crossed diplopia for distance becomes annoying. It is not amenable to non-surgical treatment.

IV. Divergence Insufficiency. This anomaly, one form of esophoria, causing the so-called "panorama asthenopia" can be benefited frequently by the prescription of prisms base out for constant use, correcting partially the esophoria manifested for distance. In the writer's experience, attempts to increase the divergence power by exercises with prisms base in are productive of no results. The result, however, of the constant wearing of prisms base out is sometimes so brilliant that it should always be tried before resorting to operative procedures.

The non-operative treatment of hyperphoria of low degree consists either in ignoring it, or in prescribing a prism base down before the eye tending to stand the higher, provided that it can be shown incontestably that the hyperphoria is still the cause of asthenopic symptoms after careful correction of the refractive error. In the writer's opinion, the bad effects of hyperphoria of low degree have been greatly exaggerated, and cases requiring operative measures after the refractive error has been properly corrected with, if necessary, the addition of a prism, are extremely rare.

THE PERCENTAGE EVALUATION OF MACULAR VISION.[1]

By ALBERT C. SNELL, M.D., and SCOTT STERLING, A.B.

AN editorial in *The Nation's Health* of January 15, 1924, discussed the need for a scientific investigation of the problem of evaluating vision. This author stated that compensation laws regarding the evaluation of total loss of vision were fairly uniform, "but when we approach the matter of compensation for partial loss of vision we find that chaos is supreme." The need for such investigation is due to the fact that many have fallen into the error of assuming that the Snellen fractional form of notating acuity expresses a percentage value of functional vision equivalent to the result obtained by the direct reduction of these notations, an assumption which, of course, leads to absurdities and confusion. This need was also felt by others long before this editorial appeared and work on the problem had already been undertaken by the Ophthalmological Section of the American Medical Association.[2] The following study was incidental to the work of that body.

The problem under consideration—that of determining the percentage evaluation of macular vision[3]—is a complex one involving physiology, physics, and mathematics, and its solu-

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[1] Paper read before the American Ophthalmological Society at Washington, D. C., May 7, 1925.

[2] See presessional volume, Section on Ophthalmology, A. M. A., 1925, report of committee on "Compensation for Eye Injuries."

[3] "Vision" throughout this paper refers only to macular visual perception.

tion depends on an understanding of the fundamental principles involved in their relationship. This solution has been arrived at as a result of the study of these underlying principles. The various steps taken in this effort were a consideration of the following points:

I. The Snellen formula.
II. Methods previously suggested for evaluating vision.
III. Usefulness of the physiological function of macular vision the basis for computing values.
IV. Investigations to determine the percentage value of fractional degrees of macular vision.
V. Application of the experimental data to the solution of our problem.
VI. Mathematical relations and computations.

I. *The Snellen Formula.*

It is obvious that retinal sensations cannot be measured but the visual angles from which the light sensations emanate can be. We measure the visual angles and thereby determine or deduce the visual acuity. The Snellen method of *measuring visual angles*, expressed by a formula, is universally regarded as scientific, practical, and the best yet devised. In it we find first, that the visual angle is *measured* by *comparing* tangents of arc, and second, these measurements are made by the use of a single standard scale. The above facts are shown from the following quotations from Prof. Snellen[1]: (Italics are ours). "Visual acuteness is *measured*—by the minimum distance at which two simultaneous impressions give rise to two independent sensations."

"We measure this visual angle by *comparing* the distance (d) at which the letters are just seen, divided by the distance (D) at which they subtend an angle of five minutes to the eye. $V = \frac{d}{D}$."

"The five minute angle has been chosen as *the standard scale.*"

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[1] *System of Diseases of the Eye*, Norris and Oliver, vol. ii, p. 11.

"The acuity of vision is expressed as the reciprocal of the smallest visual angle at which the test object can be distinctly seen."

Thus we see that every Snellen notation is purely and simply a *comparative* expression. The visual angle under which a test-object is just distinguishable is always compared with the Snellen standard in an inverse ratio. Every visual acuity test simply expresses the relation of *two* "reciprocally comparable" visual angles. In actual practice the *distance* at which the standard eye can just distinguish the standard five minute angle or test letter is always *compared* with the distance at which the five minute visual angle or test letter is just distinguishable.

The second point Snellen states clearly—that the five minute angle is a complete unit "scale"; that is, we must conceive of this five minute angle scale as being graduated at regular intervals and extending to infinity. All visual angles—all test letters—are measured by this unit scale which determines their size. Every Snellen notation is an expression of comparison between the size of the standard object and the size of the just distinguishable object whether expressed in terms of angles or of distances. Also, in the complete Snellen system of notations, each single notation expresses only the comparative relation of one measurement with another and there is no proportional, mutual relationship between different notations.

The Snellen notations have mathematical significance. Let us consider the practical application of the use of this scale in order to make clear the mathematics involved. If we choose to use integral numbers in making our comparative gradations on this angle scale, the smallest numbers in sequence would be 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, etc. to ∞. These gradations as shown above may be conceived of as expressing minutes of arc of visual angles at a fixed distance, or distance in feet of the unit angle. It must be obvious that 2 is twice as large as 1, that 3 is three times larger than 1, that 4 is four times larger than 1, etc. Computed in terms of five minutes of arc to correspond with the 5 minute standard angle scale, we obtain the following by the same method of comparison:

A	5	minute	angle	is	1	times	larger	than	the	5	minute	standard unit
"	10	"	"	"	2	"	"	"	"	5	"	"
"	15	"	"	"	3	"	"	"	"	5	"	"
"	20	"	"	"	4	"	"	"	"	5	"	"
"	25	"	"	"	5	"	"	"	"	5	"	"
"	30	"	"	"	6	"	"	"	"	5	"	"
"	35	"	"	"	7	"	"	"	"	5	"	"
"	40	"	"	"	8	"	"	"	"	5	"	"
"	45	"	"	"	9	"	"	"	"	5	"	"
"	50	"	"	"	10	"	"	"	"	5	"	"
etc.												

These comparisons might be expressed in feet and the same proportions would be found.

Using the reciprocals of these comparisons and employing the one minute angle of the scale, we would obtain the following expressions: 1, $\frac{1}{2}$, $\frac{1}{3}$, $\frac{1}{4}$, $\frac{1}{5}$, $\frac{1}{6}$, $\frac{1}{7}$, $\frac{1}{8}$, $\frac{1}{9}$, $\frac{1}{10}$, etc.—a series of fractions whose denominators are in arithmetical progression—which in mathematics is known as a harmonic progression. Applied to visual angles, or to their tangents, one cannot deduce from this harmonic series that these fractional numbers at the same time express fractional parts of macular vision; because, as we have pointed out above, each of these fractional numbers simply shows the reciprocally comparative relation of one gradation, the unit of measure in our one minute angle visual scale, compared with another, the observed visual angle. Further, these fractional numbers are not divisions of the visual unit. The one minute angle of the Snellen scale is merely the unit of comparative measure. It must be evident, then, that *these fractional numbers are not equal or aliquot parts of unity but are expressions of quantities compared with unity.*

Since some still hold to the assumption that the Snellen notations do express equivalent fractional vision, a brief consideration of this assumption is necessary to prove its fallacy. Let us consider briefly the nature of fractions. "A fraction is a part, or a number of aliquot parts of unity. Unity is regarded as divided into equal parts, and one or more of those parts as taken to constitute the fraction."[1] In common fractions a unit quantity is divided into equal parts, the denominator showing into how many parts the unit is divided,

[1] See Century Dictionary

the numerator showing the number of parts taken. Snellen notations do not express equal or aliquot parts of unity and cannot be regarded as expressing fractions of the physiological function of vision. In the first place the one minute angle is regarded as the angle which determines the standard "minimum separabile" visual angle, and is not divided into smaller parts. In the second place in the measurement of acuity by the Snellen method the one minute angle instead of being divided into parts is, in fact, repeatedly increased in size. Adding to unity or multiplying unity by any quantity cannot divide it into fractional parts. These considerations clearly show that the Snellen fractional form of expression does not express fractional vision.

Changing the Snellen notations from a common fractional form of *expression* to a decimal form, of course, does not change the principle upon which the notations are based, and does not change the value. If it is erroneous to regard the Snellen notations as expressions of fractional vision, reducing these expressions to decimal equivalents does not make the method any less erroneous.

Since the Snellen expressions or notations are not true fractional expressions of vision, quite naturally Snellen (and his immediate followers) opposed the proposition of reducing his symbols to "resultant" or to numerical equivalent fractions. Snellen also anticipated the possibility that, through misinterpretation, erroneous values might be given to his notations and accordingly gave a warning to this effect as follows[1]:

"The disadvantages which arise from the reduction of the actual equation $\frac{d}{D}$ to a *resultant* decimal are,— the arithmetical series 0.1, 0.2, 0.3, etc., tends to produce a wrong impression of the proportional values of the visual acuity which are indicated by the test."

"The reduction of this fraction to a decimal is *unfortunately* followed by many."

Monoyer, who developed the decimal notation for the dioptric system of lenses, apparently was the first to suggest the use of the decimal system of expression for acuity. But it

[1] *System of Diseases of the Eye*, Norris and Oliver, vol. ii, p. 15.

does not follow that these decimal expressions represent percentages of macular vision.

Many well known authorities among the older writers agreed with Prof. Snellen for various reasons in opposing the use of the decimal notation, or of reducing the Snellen notations to numerical resultants, among whom are Weiss, Green, E. Landolt, Fuchs and Donders.

Weiss[1] states: "I agree with those who do not express the result of the visual test in decimal fractions, but who make the numerator refer to the distance at which the test-objects subtend the visual angle of five minutes. This notation gives information as to the way in which the test has been conducted."

Green[2] points out in unmistakable language that the reduction of the Snellen notations to their arithmetical equivalents "invites or at least suggests erroneous interpretations," and leads to "erroneous and misleading evaluation" of the measured vision.

Landolt[3] shows that the Snellen notations are used in "terms of *comparison*" and that these comparative expressions are not *fractions* of vision. He shows that the decimal scale of Monoyer does not divide acuity in equal fractional parts but that each of these gradations has a ratio to the standard normal, the proportion between these gradations being unequal.

Fuchs[4] states: "The visual acuity as determined by Snellen's card is usually given without reducing the fractions," and he also states[5]: "The visual acuity, which an ametropic eye shows without glasses is its *relative visual acuity*, and *furnishes no measure whatever of the general usefulness of the eye for vision.*"

Donders[6] states, (S = acuteness of vision). "The formula $S = \frac{d}{D}$ gives neither the absolute measure of the distinguish-

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[1] Weiss, Leopold; Heidelberg, 1895. *Sehproben-Tafeln zur Bestimmung der Sehschärfe für die Ferne.*

[2] Green, *Trans. Am. Ophth. Society*, vol. x, p. 650.

[3] Landolt, *The Refraction and Accommodation of the Eye*, 1889, p. 230.

[4] Fuchs, *Text-Book of Ophthalmology*, Third Edition, p. 738.

[5] *Ibid.*, Seventh Edition, p. 182.

[6] Donders, *Accommodation and Refraction of the Eye*, 1864, p. 194.

ing power of the retina, nor *mutually comparable relative values.*" "*If an image has double the magnitude, it has not at the same time double distinctness.*" "Therefore, also, when the image must have double the magnitude to be recognized, we cannot say that the acuteness of vision is really reduced to half." It must be apparent to the reader that the Snellen notations do not have "mutually comparable relative values," because all the ratios in the Snellen series are unequal.

From the quotations given above sufficient authoritative evidence is presented to reveal and to confirm the following deductions: (1) The Snellen notations, reduced or unreduced, in the form of vulgar fractions or in the form of decimal fractions, do not express true numerical fractional parts of vision. (2) The various Snellen expressions are not mutually proportional. (3) The measurement of acuity by the Snellen scale is a relative measurement and does not measure the usefulness of an eye. (4) The Snellen fractional manner of expressing this relative relation of one quantity to another, is open to criticism in that it "invites" or "tends to give" wrong impressions of the proportional values of vision.

We wish to point out that these deductions are no criticism of the scientific accuracy of the Snellen system of expressing relative acuity. The Snellen notations are definite and their meaning is perfectly clear. The error arises only when the fractional expression of acuity is interpreted as being the equivalent fractional expression of the physiological function of vision.

II. *Methods Previously Suggested for the Evaluation of the Measured Visual Angles.*

The Snellen system is universally accepted as a standard method of designating acuity, but some have failed to comprehend the truth of the observation so clearly stated by Donders in the quotation above, that doubling the magnitude of an object does not double its distinctness; and vice versa:—When the size of an object must be double in order to be recognized by an eye, it does not follow that such an eye has lost fifty per cent. of its physiological function. This point will be elucidated later.

Recognizing, then, that the comparative notations of Snellen do not directly determine or indicate a like percentage value for the physiological function of macular vision, many authors have presented possible solutions of the problem of evaluating this function. The different methods of some of these are briefly outlined below:

John Green,[1] using the Snellen standard, made a series of test letters, which are mutually comparable by employing equal geometrical gradations, the tangent of each gradation in the series being increased by the constant 1.26. Thus all the steps in his series of test letters were made up of equal ratios, and are mutually proportional. But Green did not state the physiological visual values for these gradations.

Nicati,[2] suggested a series of visual values based on a principle which seemed to conform with Fechner's psychophysical law. He used specially constructed test types whose size increased in geometrical progression and to each such progression he computed the "physiological value" of vision in an arithmetical progression.

More recently Magnus,[3] Würdemann,[4] Hansell,[5] and Holt[6] have computed the economic value of macular vision (and the other essential factors of vision as well) on the basis of the relation of impaired vision to the earning ability of the individual. Magnus and Würdemann obtained their values by logical processes reduced to mathematical formulas which form the basis of computing the economic value of vision. Hansell computed his table largely on the basis of logic, of clinical experience, and of the effect of subnormal acuity on earning ability. Holt derived his economic values largely by processes of evolution and involution.

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[1] Green, *Trans. Am. Ophth. Society*, vol. x, p. 644. *Trans. Am. Ophth. Society*, 1868, p. 68.

[2] Nicati, *Echelles visuelles et leurs applications*, Paris, 1894.

[3] Magnus, *Visual Economics.*

[4] Würdemann, *Annals of Ophth.*, April 1901, Section on Ophth., A. M. A., 1901, *Trans. Jour. Am. Med. Ass.*, Feb. 1902.

[5] Hansell, *Annals of Ophth.*, 1900.

[6] Holt, *Am. Encycl. of Ophth.*, vol. xviii, p. 13595. Holt, *Trans. Am. Academy of Ophth. and Oto-Lar.*, 1914.

Allport,[1] Chapman,[2] Sharp,[3] Smith,[4] and others have presented tables of visual efficiency computed on the basis that only that amount of the visual angle should be considered which includes the range of economically useful vision. This angle is then divided into equal arithmetical gradations each of which is assumed to represent an equal value. This method requires that two assumptions be substantiated: First, that the point of industrial blindness is fixed, and second, that each gradation in the arithmetical series has equal useful physiological value. This cannot be done.

III. *Usefulness of the Physiological Function of Vision the Basis for Computing Values.*

The correct basis for evaluating macular vision is the measure of the efficiency or usefulness of its normal physiological function. Can this postulate be supported by authority or by logic? In support of our contention that the measure of *usefulness* of macular vision is the correct principle upon which to determine efficiency we direct attention to the quotation from Fuchs in a preceding section.[5] We direct attention also to the fact that many compensation laws, which fix a rate of loss for partial impairment of function of any human organ or part, base such percentage rates on "loss of use"[6] of such a part or organ, or on loss of "usefulness." This principle is accepted as economically correct by all students whether approached from medical, legal, judicial, or lay points of view.

From a philosophical point of view it is obvious that the value of any machine is determined by its usefulness in producing the desired results for which the machine was designed. The value of an individual to society is determined by the usefulness of such an individual in his community. The economic value of any physical force depends entirely on the

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[1] Allport, *Jour. Am. Med. Ass'n.*, 1920, lxxiv, No. 3, p. 166.

[2] Chapman, *Wis. Med. Jour.*, June 1918, xvii, p. 54.

[3] Sharp, *Am. Jour. Ophth.*, June 1920.

[4] Smith, *Trans. Am. Ophth. Society*, 1921.

[5] See reference No. 8, Sec. II.

[6] *New York State Workmen's Compensation Law*, Ed., 1922, pages 46 and 47, pp. r & s.

degree of usefulness to which such force can be employed to accomplish a desired task. Usefulness is the only basis upon which we can evaluate any force or function. Visual function is no exception. The value of macular vision should be determined by the same principles that are employed to determine the value of social relations, of physical force, or of physiological functions.

The essential physiological function of the macula is the perception of detail. By this act one object is distinguished from another, or the separate details of any given object are distinguishable. The measurement of visual efficiency,[1] then, is the measure of the ability of the physiological function of the eye, which is its power to resolve detail[2] and to distinguish separate objects within the range of direct vision.

Fortunately acuity is determined by an exact method—that of employing the Snellen method of measuring visual angles. The problem then of evaluating vision is that of determining the relation of the percentage of usefulness of this function to the measured visual angles.

The Snellen notations, which show the comparative relation of the unit visual angle with the observed visual angle, do not express percentage of usefulness, or of resolving power, as is clearly shown by the following illustrations: If one watch loses five seconds a year and another loses one second a year, the latter is five times more accurate than the former. But one cannot therefore conclude that the first watch has lost 80 per cent. of its usefulness or efficiency since both are exceptionally good watches and each should be rated high in efficiency. If an eye can see and resolve every word on this page and every letter in each word, it could not resolve the characters twice as well if the type were double the size, or four times as well if they were four times as large: And, conversely, as has been pointed out by Donders, if this print must possess double the magnitude in order to be disguished by another eye, it does not follow that the latter has only fifty per cent. useful vision. Or, to make our comparison between smaller objects which

[1] Macular visual efficiency is defined as the percentage of useful macular vision.

[2] Resolving power is defined as the ability of the eye to distinguish separate, discrete objects.

makes this fact more obvious, let us take the following example: If the right eye can distinguish clearly the $\frac{2}{10}$ Snellen test letters and the left eye can distinguish only a $\frac{2}{20}$ character, although the left eye has only one half the comparative acuity of the right, it does not follow that the left eye is fifty per cent. blind or has lost fifty per cent. useful vision.

IV. *Investigations to Determine the Percentage Values of Fractional Degrees of Vision.*

That it is necessary to make further investigation and study of the problem of determining values for macular vision, in order to establish a standard on a scientific basis, is obvious from the following considerations: (1) A few ophthalmologists still hold that the comparative measurement of visual angles expresses the percentage of vision in a simple harmonic progression. (2) The percentage of usefulness or efficiency of vision has not been determined by any method which has received universal endorsement. (3) The suggestions of Snellen, Green, Donders, Landolt and others in relation to mathematical interpretations of acuity notations were largely negative. These authors pointed out the conclusions deduced from an erroneous interpretation of the Snellen notations, but did not present any method for determining correct percentage values of vision for these notations. (4) For the solution of this problem many methods have been suggested by a large number of authors, but a uniform standard is still lacking. In order to establish a standard for evaluating vision[1] the American Medical Association appointed a committee to study this complex problem. After several years of investigation their report has now been presented.

The writers of this paper working independently but with

[1] In general, compensation statutes fix the basis for ocular damage on "loss of sight," "loss of vision" or "reduction of vision." See statutes of Pa., N. Y., Tenn., Vt., Ind., Me., Conn., etc. The statutes do not mention relative or comparative visual *acuity* as the basis for computing compensation. The New York statute reads: "Compensation for loss of binocular *vision* or for eighty per centum or more of the *vision* of an eye shall be the same as for loss of the eye." "The Department takes only direct *vision* into account" (*New York State Workmen's Compensation Law*, August, 1922, p. 45.)

the purpose of assisting the committee of the American Medical Association in its investigations studied the problem from three different points of view, clinical, experimental, and mathematical.[1]

Clinical data. A survey of the clinical data has been presented in a recent paper prepared for the American Medical Association[2] to which the reader is referred. A perusal of this data shows that no loss to earning ability was found until acuity was less than $\frac{20}{60}$ Sn. in the better eye; although such a substandard degree of acuity does cause some loss in efficiency and has an economic bearing on the competitive ability of workers. For this reason in evaluating vision one must not omit the consideration of the accumulative effect of repeated trivial losses of acuity. From a purely clinical point of view a $\frac{20}{40}$ Sn. notation cannot be regarded as expressing one half loss of earning ability or a loss of one half of the usefulness of an eye, since no person with a $\frac{20}{40}$ Sn. acuity was found to have lost either one half of his efficiency or of his earning ability. In fact a $\frac{20}{40}$ Sn. acuity was found to cause no loss to earning ability and only a slight loss to efficiency.

An acuity of $\frac{20}{400}$ Sn. was found to be a total incapacity for work for the majority of cases, yet some were found with working capacity whose acuity was $\frac{5}{200}$ and $\frac{3}{200}$ Sn.

Experimental investigations. As a basis for our experimental investigation we kept in mind that the problem was that of evaluating subnormal vision in terms of *percentage of efficiency*, or usefulness. We therefore laid down the following premise; that the entire range of quantitative acuity must be considered, and a scale determined, such that each gradation in the scale would have a definite value in terms of usefulness. No previous scale could be found which fulfilled the requirements

[1] The clinical data were compiled and contributed by Dr. Snell. The idea for the experiments was first conceived by Dr. Snell and the experiments were performed by him individually. Mr. Sterling modified the method and made independent experiments. Some experiments were made in coöperation. The mathematical relations and computation are Mr. Sterling's individual contributions. The principles involved, the conclusions, and their application to this problem are the result of our combined experience and study.

[2] See presessional vol. Section of Ophthalmology, A. M. A., 1925, "Visual Efficiency of Various Degrees of Visual Acuity," etc.

of this premise, and we were obliged to approach the problem by some new and different method.

It occurred to us that if we could produce an artificial condition, which in its effect on vision, would closely simulate that of a lesion, we could by experiment and measurement determine the effect of such a lesion on different degrees of acuity, normal or subnormal. It seemed obvious that an obscurant glass that was partially opaque, or covered with scratches, when placed before the eye, would reduce the visual acuity by a measurable amount. Upon experiment it was found possible to make an obscurant which had an effect on acuity quite similar to that of a corneal opacity or to disturbances of the media.

At this point, several questions relating to the value of our experimental data had to be carefully considered. Among the more important of these were:

(1) To what extent would the human factor or personal element cause variations in the subjective observations with the use of the same obscurant?

(2) Could an obscurant with a constant effect on vision be found, so that it might be standardized?

(3) With different eyes of equal acuity, either normal or subnormal, would the same obscurant reduce the acuity by a definite amount?

(4) When identical obscurants were used in multiples—two, three or more times—would the increase in the visual angle be constant for each obscurant added?

Without answering all of these questions in detail at the present time, suffice it to say here[1] that several different kinds of obscurants were tried. Among them were photographic films having different degrees of density, and glasses with various amounts of opacity made by grinding, sand-blasting, or etching. All of these were found to be unsatisfactory for various reasons. The most satisfactory obscurant was a meshed glass made by cutting into the surface of the glass, fine lines close together, and crossing each other at different angles. The lines were made as uniform as possible, both as

[1] The complete details of various methods employed in these experiments, together with complete data, results, and conclusions will be published later.

to depth of cut and spacing between the lines. These meshed glasses were not made all alike—dissimilar patterns were selected, to simulate the purely accidental effect of eye lesions, and to preclude the possibility of the lines of one obscurant falling directly before corresponding lines of a second glass, when the two were used in multiple.

A number of meshed glasses were used which had the property when placed before the eye, of reducing the acuity by a definite amount. Some reduced the acuity from $\frac{20}{20}$ Sn. to $\frac{20}{30}$ Sn. Some reduced the acuity from $\frac{20}{20}$ Sn. to $\frac{20}{400}$ Sn. or more. Several glasses were selected which reduced the acuity from $\frac{20}{30}$ Sn. to $\frac{20}{40}$ Sn. These $\frac{20}{40}$ glasses were standardized by testing a large number of patients. A number of other meshed glasses which caused a greater decrease of acuity were standardized in the same manner, but the $\frac{20}{40}$ meshed glass was taken as a unit or standard because it increased the visual angle of one minute by one minute. With the $\frac{20}{40}$ meshed glass before the eye, the standard 5 minute character had to be increased to 10 minutes to be seen by the normal eye, an increase of 5 minutes in the size of the test character.

We then proceeded with our experiments in a threefold manner:

(1) The same obscurant or its equivalent was used in series, two, three, four or more meshed glasses being placed before the same eye.

(2) The standard or unit meshed glass was placed before different eyes with subnormal acuity.

(3) The acuity of an eye was first reduced by means of spherical lenses, and then the effect of adding one or more of the standardized meshed glasses was determined.

Space in this paper permits only a brief summary of the results of our experments:

(a) In so far as the experimental data is concerned, the personal element was found to be practically the same whether meshed glasses were used or not. The same factors that influence visual acuity determinations under ordinary subjective conditions were encountered in our experiments.

(b) The effect of the standard meshed glass on acuity was found to be constant and variations were slight until acuity

fell below $\frac{3}{200}$ Sn. A given meshed glass was found to produce a definite increase in the visual angle throughout the range of visual acuity from $\frac{3}{10}$ Sn. or better to $\frac{3}{200}$ Sn.

(c) When six of the $\frac{3}{10}$ unit meshed glasses, or their equivalent, were used in series before the eye it was found that the acuity was reduced to a point approaching qualitative sight.

V. *Application of the Experimental Data to the Solution of our Problem.*

From these observations and from a consideration of Weber's and of Fechner's psycho-physical laws, and of Lambert's law of light absorption, the following mathematical deductions have been drawn. An obscurant glass increases the visual angle, and in so doing takes away some proportional part of the visual efficiency or usefulness of the eye. Since the amount of increase in the visual angle is constant for a given obscurant, *equal increases in the visual angle correspond to equal proportionate reductions in the visual efficiency*

In other words, *the visual efficiency of an eye diminishes in a constant ratio for successive equal increases in the visual angle.* Another way of expressing the same relationship is: *Equal increases in the visual angle reduce the visual efficiency by a constant proportion.*

To illustrate this idea let us suppose that we have a number of cases all with different amounts of useful vision—the first has a visual efficiency of 100%; the second 50%, and the third 10%. Let us further suppose that we place an obscurant that reduces the visual efficiency 20%, before each of these eyes. The visual efficiency of each eye will be reduced 20% of its value by the obscurant—the 100% eye will be reduced to 80%; the 50% eye will be reduced from 50% to 40%; and the 10% eye from 10% to 8% visual efficiency.

The same obscurant produces the same percentage of loss of visual efficiency in each case, and each eye loses exactly in proportion to its value. The normal, or 100% eye loses twice as much as the 50% eye, and ten times as much as the 10% eye.

This same fact is stated in mathematical terms as follows: *As the visual angle increases in an arithmetical ratio, the visual*

efficiency diminishes in a geometrical ratio. This expression is one for which a mathematical equation can be given, and this equation can then be solved by regular mathematical methods.[1]

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[1] The complete development of the mathematical relationship is set forth in the following computation.

Visual angle = a = the minimum angle subtended at the eye by two objects that can be distinguished as separate.

Normal visual angle = a_N = one minute of arc.

Visual acuity = 1/a = the reciprocal of the visual angle in minutes of arc.

Snellen notation = the reciprocal of the visual angle in minutes of arc, expressed in fractional form with the examining distance as the numerator.

Visual resolving power = R = the measure of the ability of the eye to distinguish separate, discrete objects before the eye.

Normal visual resolving power = R_N = the measure of the ability of the normal eye to distinguish as separate two objects subtending the normal visual angle of one minute.

Visual efficiency = E = "vision" = the ratio of the visual resolving power of an eye to the visual resolving power of the normal eye. Visual efficiency is expressed as a fraction, or as a percentage.

$$E = \frac{R}{R_N}$$

hence $$R = R_N.E \qquad (\textit{Equation I})$$

Loss of vision = 1.00 − E = the loss of visual efficiency.

The relation between the visual angle and the resolving power is expressed by the following: *As the visual angle increases in an arithmetical ratio the resolving power diminishes in a geometrical ratio.* The equation for this relation is:

$$e^{-ka} = R_N.E \qquad (\textit{Equation II})$$

where e is the base of natural logarithms, 2.71828; k is a constant; R_N is the resolving power of the normal eye; and E is the visual efficiency.

Equation II reduces to the expression

$$-ka \log e = \log R_N + \log E$$

but $$k \log e = K = \text{a new constant}$$

hence $$Ka = \operatorname{colog} R_N + \operatorname{colog} E \qquad (\textit{Equation III})$$

For a visual acuity of $\frac{10}{10}$

$$a = 1.0,\ E = 1.00, \text{ and } \operatorname{colog} E = 0$$
$$K = \operatorname{colog} R_N$$

Substituting the value K for colog R_N in Equation III

$$Ka = K + \operatorname{colog} E$$
$$K(a-1) = \operatorname{colog} E$$

or $$K = \frac{\operatorname{colog} E}{a-1} \qquad (\textit{Equation IV})$$

VI. *Mathematical Relations and Computations.*

The visual efficiency values corresponding to every possible visual angle can be determined from the formulæ when two definite values have been established. For the first of these we have taken normal, or $\frac{20}{20}$ Sn. as 100% efficiency. Although many visual economists have held that there is no loss of useful vision until $\frac{20}{30}$ or $\frac{20}{40}$ has been reached, we have accepted for our first value the standard one minute visual angle (five minutes for the size of the entire test letter), as laid down by Snellen and used by ophthalmologists the world over.

Since six of the $\frac{20}{40}$ unit meshed glasses in series before an eye reduce acuity to a point approaching qualitative vision, the reduction in visual efficiency caused by one of these obscurant glasses is approximately one sixth of the whole, or about 16%. On the basis of 16% loss of visual efficiency for $\frac{20}{40}$ Sn. the loss of visual efficiency is approximately 50% for $\frac{20}{100}$, and 80% for $\frac{20}{200}$. These are exactly the values laid down by Hansell,[1] and Magnus.[2] While Hansell's table differs from ours in that Hansell did not consider $\frac{20}{30}$ Sn. or even $\frac{20}{40}$ Sn. as representing any loss whatever of useful vision, it is inter-

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For a visual acuity of $\frac{20}{200}$

$$a = 10.0 \qquad E = 0.20, \text{ and colog } E = 0.6989700$$

$$K = \frac{0.6989700}{10-1} = \frac{0.6989700}{9} = 0.0776633 = \text{colog } R_N = -\log R_N$$

$$\log R_N = 9.9223367$$

from which we have the important relationship

$$R_N = 0.83625 \qquad (\textit{Equation V})$$

R_N denotes not only the visual resolving power of the normal eye, but also the constant ratio of decrease in visual efficiency for each increase of one minute in the visual angle.

In other words, since $R_N = 0.83625$, *each increase of one minute in the visual angle reduces the visual efficiency to the 0.83625 part of what it was before such increase.*

Expressed in the simplest form, the equation for this relation is:

$$E = R_N^{(a-1)} = 0.83625^{(a-1)} \qquad (\textit{Equation VI})$$

[1] Hansell, *Annals of Ophthalmology*, vol. ix, No. 4, p. 605.

[2] Magnus, *Visual Economics*, p. 105.

esting to note that in the lower acuities, the percentage values of useful vision in the Hansell table correspond almost exactly with our own, although the two tables were developed by entirely independent methods. In our table $\frac{20}{200}$ Sn. is taken as representing 80% loss of visual efficiency—exactly the same value as that given by Hansell and Magnus. $\frac{20}{100}$ Sn. in our table corresponds to 51% loss of vision, while according to Hansell it represents 50% loss.

SUMMARY.

The Snellen scale measures the visual angle and the Snellen notation for visual acuity expresses the reciprocal of the visual angle in fractional form.

This fractional expression is not the measure of the percentage value of fractional degrees of macular vision.

The basis for evaluating macular vision is the measure of the ability of the eye to perform its normal physiological function, which is the perception of detail.

Experimental investigations were made with meshed glasses to produce an artificial condition simulating the effect of ocular lesions or defects. A meshed glass which had the property of increasing the visual angle of one minute by one minute, or the standard five minute character by five minutes, was taken as the standard obscurant. This is equivalent to a reduction of acuity from $\frac{20}{10}$ to $\frac{20}{40}$ Snellen.

The standard meshed glass was found to produce a definite increase in the visual angle throughout the range of quantitative vision and six of these in series reduced acuity from $\frac{20}{10}$ Snellen to a point approaching qualitative vision.

From our experimental observations and from a consideration of physical and psycho-physical laws the following mathematical deductions have been drawn:

The visual efficiency of an eye diminishes in a constant ratio for successive equal increases in the visual angle. In other words, as the visual angle increases in an arithmetical ratio, the visual efficiency decreases in a geometrical ratio.

From these deductions we find that each increase of one minute in the visual angle reduces the visual efficiency of an eye to the 0.83625 part of what it was before such increase.

The table based on this constant shows the Snellen acuity notations and the corresponding visual efficiency values for the entire range of quantitative vision.

RELATION BETWEEN VISUAL ACUITY AND VISUAL EFFICIENCY.

By A. C. Snell and Scott Sterling.

Snellen Notation	*Visual Angle in Minutes*	*Visual Efficiency in Percent*	*Percentage Loss of Vision*
20/20	1.0	100.0	0.
20/25	1.25	95.6	4.4
20/30	1.50	91.4	8.6
20/40	2.	83.6	16.4
20/50	2.5	76.5	23.5
20/60	3.	69.9	30.1
20/70	3.5	63.8	36.2
20/80	4.	58.5	41.5
20/90	4.5	53.4	46.4
20/100	5.	48.9	51.1
20/120	6.	40.9	59.1
20/140	7.	34.2	65.8
20/160	8.	28.6	71.4
20/180	9.	23.9	76.1
20/200	10.	20.0	80.0
20/220	11.	16.7	83.3
20/240	12.	14.0	86.0
20/260	13.	11.7	87.3
20/280	14.	9.8	90.2
20/300	15.	8.2	91.8
20/340	17.	5.7	94.3
20/380	19.	4.0	96.0
20/400	20.	3.3	96.7
20/500	25.	1.1	98.9
20/600	30.	0.6	99.4
20/800	40.	0.1	99.9

SYPHILIS AND IRITIS.

By Dr. H. DICKSON BRUNS, New Orleans, La.

IN 1916 at the meeting of the American Ophthalmological Society, I listened with interest to a paper presented by Dr. Ernest E. Irons and Dr. E. V. L. Brown reporting a painstaking examination into the causes of one hundred cases of acute iritis seen in Chicago. Later, Dr. Brown kindly sent me a reprint of this paper together with a second report on one hundred cases by Dr. Irons and himself, read before the section on Ophthalmology of the American Medical Association at the meeting in San Francisco, June, 1923.

In reading these papers, while admiring the thoroughness of the investigation, I was struck by the small number of cases ascribed to syphilis. My impression was deep that here, at the southern end of the Mississippi Valley, the majority of our patients with acute iritis are also victims of the specific disease. Is it possible, I have often asked myself, that in so vast a country as the United States the prevalent causes and the course of diseases may be somewhat different in widely separated areas? Or do variations arise solely from the different racial stocks preponderating, the densities of population and the unlike conditions such differences entail?

I determined to find out the incidence of syphilis in cases of acute iritis seen in our clinic as shown by the Wassermann test. It was not until the years 1920–21 that, aided by Dr. Kotz Allen, of the visiting staff of the Eye, Ear, Nose and Throat Hospital, I was able to attend to the clinical side of this comparatively simple investigation. To Dr. Allen is owing not only clinical assistance, but all the clerical work. Indeed his zeal led him to look over the hospital's records of iritis cases back to 1915.

In 1920 we treated in the clinic 109 cases; white males 19, white females 7, colored males 45, colored females 38 cases. The ages ran from 10 to 71 years, with an average of 35 years. Four (4) cases were recurrences or relapses. Treatment was begun from one day to 6 years—doubtless a relapse—after the onset of the disease; it lasted from one day to 7 months with an average of 22 days. The one day cases, 25 in number, were those who never returned after their first visit. Wassermann tests were made in 77 cases. They were reported positive in 59, or 76+%, more than 1+ in 46 instances. The test was negative 18 times, or 23+%. For making these tests I am obliged to Dr. W. H. Seemann, the pathologist to the hospital, who used both the cholesterin and the Noguchi method.

In 1921 we saw 93 cases; white males 16, white females 2, colored males 48, colored females 27 cases. The ages were from 11 to 65 years, with an average of 25 years. Treatment was begun from the first day to the 24th month, with an average of 7 days, after the onset of the disease. It was continued from one day—the patient never returning—to 12 months, with an average of 21 days. Thirty-one (31) of these patients were never seen after their first visit; a Wassermann test was made by Dr. Seemann in all the other 62 cases. In 35, or 56+%, the test was positive, only 3 under 2+, and in 27, or 43+%, cases negative. Eleven (11) cases are recorded as traumatic. This unusual excess of traumatic cases —over 40% of the "negative Wassermanns"—helps to account for the small percentage of positive findings in this 1921 group of 93 cases. For if we go back to the records of 1919, when 121 cases were treated and 63 Wassermann tests made, we find 48, or 76+% (again) positive, only 5 being less than 2+. If we add these 63 cases to the 62 of 1921 and the 77 of 1920, we get 202 in which Wassermann tests were made, of which 142, or almost exactly 70%, were positive; of these only 5 were less than 2+.

With the exception of the 1919 cases, the Wassermann tests made were too few to be of value, but since we have these reports before us there are certain facts about the 518 cases of acute iritis that seem noteworthy, though not entirely pertinent.

The average age of the patients in the 1916–17 group of 111 was 22 years; of the 84 cases of the 1918 group, 24 years; of the 121 cases of the 1919 group, 25 years; of the 109 cases of the 1920 group, 30 years; of the 93 cases of 1921, 25 years. Confirming the accepted belief that acute iritis is a disease of early adult life. Males were much more numerous, 325 (78 white, 247 colored) to 193 (22 white, 171 colored) females. Figures which also show the colored patients, 418, to have greatly exceeded the whites, 100.

This large proportion of colored persons among our patients might be held to explain the apparent preponderance of a specific cause for the cases of acute iritis. The permeation of this race as we see it in our southern clinics with syphilis is well known. It might be argued, therefore, that a specific basis might be ascribed to any disease to which the race is subject. The percentage of persons of color, however, among all who attend our clinic has never exceeded 40; a clear indication that this highly syphilized group is especially liable to iritis, *i.e.*, that the two diseases are, at any rate, likely to coexist. It cannot be denied that an iritis in a syphilitic might be due to some other cause, but in the presence of a frankly positive Wassermann reaction I should regard it as syphilitic until there was plain proof to the contrary.

Of the 518 cases, 183 never returned after the first visit; 243 quit improved shortly before they would have been discharged, conduct familiar to all who have worked in free clinics. Ninety-two (92) were discharged cured.

In the five groups into which the 518 cases were divided, including the 92 discharged cured and the 243 who quit improved, the average duration of treatment was, respectively, 22, 21, 20, 22 and 21 days. Had a greater number waited a few days to be discharged, I believe the figure for the average duration of treatment would have been nearer to 28 or 30 days. This has been the figure I have found for a number of groups of 100 cases reviewed from time to time. This point has been particularly interesting to me as indicating the correctness of a view I have long entertained,[1] that

[1] "Bruns Acute Plastic Iritis." A paper read before the Louisiana State Medical Society; annual meeting, 1894—Published in the *New Orleans Medical & Surgical Journal* of that year.

acute iritis is, with certain qualifications, a self-limited disease. These qualifications are, that the case come under treatment with reasonable promptness, that the pupil be kept fully dilated and that no serious complications arise, *i.e.*, many unbreakable synechiæ, severe and persistent extension backward along the uvea. By this I mean that, these conditions being fulfilled, in any large group of cases the average time from the beginning of treatment to the discharge of the case as cured, or practically cured, will be found to be from 20 days to a month regardless of any other treatment administered. The very short and the unduly prolonged cases being more than compensated for by those running the 20 or 30 day course. The cases here considered, besides the atropin and bathing of the closed eye with very hot water, were treated by inunctions of "Arlt's Salve" (ung. hydg. ℥j, ext. bellad. ℥j, divided into 9 portions) one portion rubbed in for 30 minutes; in very resistant cases even twice daily,[1] with hot or Turkish baths daily when possible. When the eye was practically well, as many as could be induced to go were sent to the genito-urinary clinic for intravenous treatment.

Finally it may be noted that in the five groups considered gummata or condylomata of the iris occurred in 5+%, 6+%, 4+%, 4+% and 5+%.

[1] Holding to the time-honored belief that we have in mercury, whenever not specially contraindicated, the drug generally most valuable in allaying inflammation and in promoting resorption of exudates I have used it always in the treatment of iritis, and indeed in all uveal inflammations, whether regarded as specific or not, lc. p. 66. In inflammation of the uvea especially, it rarely disappoints one. We are assured of how preposterous it would be to suppose a syphilitic infection could be eliminated during the usual course of an acute iritis by seeing the second eye light up during active anti-specific treatment. Now that we know a negative Wassermann report to be worth little more than the paper it is written on, we cannot but wonder how many of these cases would have proved to be positive could we have had spinal fluid examinations.

A PLEA FOR REVIVING THE OPERATION OF COUCHING.

By Dr. G. LINDSAY JOHNSON, Durban, Natal, So. Africa.

THIS operation, probably the oldest method of operating for cataract in existence, has been practised for centuries in India and China. It is a delightfully simple affair. It consists in thrusting a needle through the margin of the cornea into the anterior chamber in front of the iris, and pushing it between the iris and the cornea on the opposite side until it almost touches the ciliary body, and then forcibly depressing the point until the lens is pushed down into the virteous completely out of the pupillary area. It usually sinks down until it rests at the bottom of the retina, but it may hang suspended by some of the unbroken fibers of the ligament. Nothing further is required. The patient sees at once and can be fitted with glasses the next day.

This operation possesses many advantages. It is bloodless. It causes no pain, and there are no immediate complications. On the other hand it has one ***most serious*** objection. It has been abundantly proved that the lens acts as a foreign body, and will inevitably lead to total blindness in the course of one, two, or at the most three years, and in rare cases after a few months, or even weeks. This is the chief reason why it is never recommended by ophthalmic surgeons at the present day.

The reader will naturally ask why I should recommend it. My reasons are as follows. I have on six separate occasions after performing what seemed to me a perfectly executed operation by removal of the lens by the classical modified Graefe operation, or else by removing the lens in its capsule, had the most disastrous sequelæ. This latter operation I

invariably perform whenever possible, as it is in my opinion an ideal operation, and in my hands has never been followed by any serious loss of vitreous, and in the great majority of cases by none at all, or the merest bead of humor.

Now one is often called upon to operate on old people suffering from arterio-sclerosis, or with diseased choroidal vessels, accompanied by high arterial (not ocular) tension. In such cases the operation will in all probability succeed perfectly, and the surgeon will go away assuring the patient that he will recover his sight. But sometimes as has happened to myself on three occasions, the lens instead of coming out of the chamber slowly and with some difficulty, has almost shot out of the eye, apparently of its own accord, before the slightest pressure has been made on the cornea, or even before a discision has been made in the capsule. This has invariably been due to a rupture of one of the choroidal vessels, and has been immediately followed by the entire contents of the eye being pushed out of the wound. Vitreous, retina and choroid appeared, followed by the most prolific hemorrhage, the whole forming a horrid blackish mass of tissues and clotted blood, which was only prevented from entirely escaping by the attachment of the retina to the optic nerve stalk.

On one occasion the bleeding was so violent that it was not merely a question of the patient losing his sight, but his life, and I was compelled to excise the eye immediately, and even then I failed to control the bleeding until I had applied the actual cautery. Lately, I have had the misfortune to have three cases one after another, in which the same disaster followed, the bleeding coming on about half an hour later, and on arriving at the bedside, I found the bandages and pillows saturated with blood, and it was with the greatest difficulty that I was enabled by applying pressure to stop the hemorrhage.

The object of this paper is to point out that when called upon to try and recover some sight in the remaining eye of an old patient in whom hemorrhage of the choroidal vessels has occurred, the operation of couching is not only justifiable but right and in fact, the only thing to do. Although we know that the eye will eventually become quite blind, nevertheless the patient will still have a certain amount of useful

sight for one if not for two years, whereas if left alone he would at the most merely be able to distinguish light from darkness. My experience extending over very many years has taught me that whenever there are signs of diseased vessels, high arterial tension, or the appearance of a general breaking up of the constitution, the surgeon should consider well before operating in the ordinary manner, even if the first eye has not been touched, and should seriously consider whether the advantages of couching do not outweigh those of the more usual methods of treatment. Lastly the surgeon must not forget that in such broken down old people, a hemorrhage of the eye will have such an injurious effect on their minds, that it will in all probability greatly accelerate their death, which is an additional reason for pausing before opening the eye in the usual way.

OPHTHALMIC DISORDERS OF ENDOCRINE ORIGIN IN WOMEN.[1]

By Dr. O. R. Lourie, Boston, Mass.

IT is well known that visual disturbances occur in women during puberty, menopause, amenorrhea, suppressed menses, dysmenorrhea, pregnancy, and lactation.

The causes were looked for previously in:

1. High blood pressure, partly in cases of temporary or permanent cessation of menses, resulting in hemorrhages either into the optic nerves or into the membranes of the eyeball, or even in the brain.
2. Circulatory abnormalities.
3. Functional changes in the nervous system.

In his book *Uterus und Auge*, Cohn (1) gathered a great deal of material on this subject. He noticed that some of the cases had not offered sufficient explanation. It was evident that they needed further investigation.

The study of the glands of internal secretion and X-ray examination of the sella turcica has thrown some light on the matter, but leaves a great field for research.

About eight years ago I read a paper before the Kiev Medical Society on four cases of tumors of the hypophysis cerebri without acromegaly, attended by visual disturbances and sexual changes. In every case we had observed bitemporal hemianopsia and atrophy of the optic nerve. A few analogous cases came to my attention later on. The diagnosis had been confirmed by X-ray, which showed enlargement of the sella turcica in every case, and sometimes absorption of the pro-

[1] Read before New England Ophthalmological Society, March 20, 1923.

cessus clinoidei postici and even of the dorsum of the sella turcica.

I shall cite a few of my cases briefly.

1. Man, 49. Loss of beard and of pubic and auxiliary hair. High pitched voice. Impotency. Headaches. Complete loss of temporal field and central vision of right eye. Complete loss of temporal field of left eye and decrease of its central vision. Ophthalmoscopic findings: atrophy of optic nerves. Diagnosis: Hemianopsia bitemporalis. Atrophy of optic nerves. Tumor of hypophysis cerebri. X-ray: Antero-posterior enlargement of sella turcica.

2. Woman, 40. Had never menstruated. Infantile uterus. Headaches. Apathy. Hair of armpits and pubes falling out. Obesity. Sexual apathy. For the last years noticed visual disturbances. Complete bitemporal hemianopsia. Acuity of central vision at that time $\frac{1}{10}$ in left eye and $\frac{6}{10}$ in the right one. Ophthalmoscopic examination showed optic atrophy. X-ray: Enlargement of sella turcica. Diagnosis: Tumor of hypophysis cerebri.

3. Girl, 23. Came for refraction as she noticed a diminution of vision. Obesity. Hair on face. Bitemporal hemianopsia. Almost blind in her left eye; decrease of the central vision of the right one. Ophthalmoscopic examination: Atrophy of optic nerves. (Had never menstruated and had never consulted any physician.) X-ray: Enlargement of sella turcica. Diagnosis: Tumor of hypophysis cerebri.

These cases of atrophy of the optic nerves as a result of pressure produced by an enlarged hypophysis upon the chiasm in conjunction with the general picture of the illness, indicate very definitely the connection between an enlarged pituitary gland and sexual mal-development on one hand, and visual abnormalities on the other. Just which is the cause and which is the result is difficult to state: an enlargement of hypophysis may cause wasting of genital organs, and on the contrary, a mal-development of the latter could be the cause of an hypophysial enlargement. Erdheim and Stumme (2) have proven experimentally that in pregnancy the hypophysis enlarges as a result of disturbed correlation of the secretory glands and that morphological changes take place in the pituitary body as the appearance of so called "cells of gestation." The following quotation is from this authority.

"During pregnancy the hypophysis undergoes a remarkable increase in weight. The minimum weight of 65 cg. in a primipara is larger than the minimum weight in a nullipara, even the maximum known weight of 75 cg. in a nullipara does not equal the average figure of 84.7 cg. in the primipara. At the termination of pregnancy the weight is again reduced, but should pregnancy again occur it rises to a figure even higher than before. The average weight of the hypophysis in the multipara at the termination of normal pregnancy is 106 cg.—a figure considerably in advance of the weights which have been ascertained in the case of the primipara. The difference in the maximal weights is even greater than that of the multipara, being 165 cg. and that of the primipara 110. The increasing weight is accompanied by structural changes."

I wish to note here that the first scientists who published articles about the hypophysis were Russians: Prof. Peremeshko and Dr. Rogowitch. The latter showed that the removal of the thyroid gland is the cause of hypertrophy of hypophysis and therefore stated the inter-relation between the glands of internal secretion.

The above mentioned facts urged me to attempt to find a more satisfactory solution of the causes of ophthalmic disorders in women during puberty, climax, amenorrhea, dysmenorrhea, suppressed menses, pregnancy and lactation. Unbalanced correlation of the internal secretory glands and changes in the pituitary body form the new starting point for attention and research.

A few years ago I read a paper before the Kiev Physico Medical Society on this subject, in which I pointed out that the etiology of ophthalmic disorders in these cases should be looked for in the compensatory enlargement of the hypophysis and in the toxins resulting from the disturbed correlation of the endocrine glands. Professor Finlay (3) in his paper "Bitemporal Contraction of Visual Fields in Pregnancy" cites a series of extremely interesting observations which have led him to the following conclusions:

"From an examination of these visual fields we find that out of the 31 cases examined, only 9 could be considered approximately normal, the remainder showing changes in

the nature of a temporal contraction; of these 8 were slight, 9 moderate, and 5 pronounced. Those surprising results led me to a thorough study of the literature at hand"; and further:

"I eventually discovered that changes in the visual fields of a similar nature to those which I have recorded have been published, first by Bellinzona and Tridonani (*Boll. d. soc. med.-chir. di Pavia*, 1903) and later confirmed by Forti (*Arch. di ottal.*, February, 1910), which they all attributed to dynamic or vasomotor disturbances similar to those occurring in hysteria.

"This more or less forced explanation seems to me unnecessary, as the changes described can be perfectly explained by the mechanical pressure brought to bear on the chiasm by the enlarged pituitary gland resulting from the normal hypertrophy of this organ which occurs in pregnancy." Further:

"In conclusion I consider I can establish: That during pregnancy there often occurs as a result of the normal hypertrophy of the pituitary gland in connection with this state, a compression of the chiasm, which manifests itself by changes in the visual fields, in the nature of a bitemporal contraction which varies in degree according to the amount of compression suffered, this depending on the corresponding amount of hypertrophy and on the anatomic peculiarities of the case which may favor or hinder this compression."

It gave me great pleasure to read Finlay's report, as he confirms my opinion on this subject expressed several years ago. But I had in mind not only pregnancy but also various visual disturbances of genital origin, as under these conditions I think there takes place hypertrophy of the hypophysis as a result of hyposecretion of the ovaries. Many cases of bitemporal or monotemporal hemianopsia may be explained by the mechanical pressure brought to bear on the chiasm. Cases of atrophy of optic nerves without typical hemianopsia may be also explained in the same way as it has been proven that enlargement of the hypophysis may ensue in the absence of hemianopsia. This is the reason why, in some of Prof. Finlay's cases, there was no contraction of visual field. Sudden amaurosis following labor or cessation of menses and disappearing afterwards, headaches, œdema papillarum and other appearances of high intracranial pressure find their etiology in a hypertrophied hypophysis.

It is more difficult to explain inflammatory processes in the optic nerve and other structures of the eye. In these cases we may look for causes in the toxins created in the blood system as a result of disturbed endocrine interrelation. The old belief that disturbed circulation, chiefly venous statis usually of reflex origin leads to absorption of lymph which irritates the optic nerve and the eye membranes,—must be modified by the addition of "through the presence of the above mentioned toxins."

Cohn in his book, relating a case of blindness which occurred two months after amenorrhea had started, says that the ordinary explanation does not seem to satisfy him as blindness set in rather late for the occasion, and that there must be some factor acting upon the walls of the blood vessels, and in this way causing rupture or permeability of the latter. But in unbalanced endocrinal correlation accumulation of toxins takes place gradually, and the effect of it is discovered after the blood becomes more or less saturated. It is extremely difficult to think of all the cases of iritis, choroiditis, neuroretinitis, and keratitis resulting from circulatory disturbances. We infer that unbalanced inter-relation of the endocrines will, if it reaches a definite stage cause a considerable hypertrophy of the hypophysis or accumulation of toxins in the blood, or both at the same time. However, circulatory disturbances may be a contributory factor. Thus we can explain all ophthalmic disorders observed in such cases.

It is also probable that nephritis gravidarum causing neuroretinitis and accompanied by blindness, also eclampsia, are due to disorders of the glands of internal secretion.

I shall quote from Cohn's book some of the cases of ophthalmic disorders in women, described in the literature. I shall take the privilege of mentioning a few of my own cases observed during my twenty-five year's practice at Kiev hospitals and clinics. I do not think that for my purpose it is necessary to quote all observations in the literature, as the character of the ophthalmic disorders in all the cases is more or less the same.

For convenience, I shall group all the ophthalmic disorders in the following manner:

A. Contraction of the visual field
B. Œdema papillarum
C. Atrophy of optic nerve
D. Neuritis optica
E. Neuroretinitis
F. Amaurosis and amblyopia
G. Hemorrhages
H. Iritis, choroiditis, keratitis, diseases of eyelids.

A. Changes in the visual field were observed in normal menstruation, in dysmenorrhœa, and in pregnancy.

Summing up the contractions of the visual field of genital origin, we have to conclude that they may be concentric, bitemporal, sometimes homonymous bilateral, monocular. None of the mentioned cases showed complete bitemporal contraction of the visual field as was observed in the cases of tumor of the hypophysis with dystrophia adiposo-genitalis.

B. Œdema papillæ nervi optici.

Leber (4) observed one case of œdema in a woman during normal menstruation and another after third delivery. I saw several cases of œdema papillarum and diminution of central vision in girls before the first menstruation, and in women undergoing climacteric changes. The diagnosis of those cases can be mistaken for cerebral tumor.

C. Atrophy of the optic nerves.

Leber (4) describes a case of atrophy in a girl who being physically developed did not menstruate.

Galezowski (5)—a case of atrophy during sudden cessation of menstruation. I personally observed a case of atrophy in a woman of 19, primipara, in the eighth month of pregnancy.

Christensen (6) described a case of epileptic amaurosis following cessation of menses which resulted in atrophy of the optic nerves.

D. and E. Neuritis optica, neuritis retrobulbaris, neuroretinitis.

Such cases were described as having taken place during climacteric, dysmenorrhea, and other disturbances of menstruation by Galezowski (7), Mooren (8), Meyer (9), and others.

Retinitis gravidarum does not differ from retinitis in Bright's disease. It appears in the sixth month of pregnancy

or earlier. Sometimes an atrophy of the optic nerve develops while the retinal changes disappear. Papilla is white and the arteries narrow.

F. Amaurosis and amblyopia.

These cases were observed as preceding first menses, in pregnancy, shortly before, during or after delivery, during lactation, in delayed menses, suppressed menses—Pechlinus (10), Brierre de Boismont (11), Bartisch (12), Morgagni (13), Frank (14), Skorkowski and Kofminski (15), Taylor (16). Blindness in these cases is generally but not always bilateral, and disappears as soon as the cause of it is over.

So called "uremic amaurosis" develops during pregnancy within six or twelve hours or even suddenly. Albumin may be absent.

Foerster (17) quoted a remarkable observation by Gibbon where a woman fattened through three successive lactation periods and remained very weak muscularly. Amblyopia was pronounced each time and resulted in blindness during the third lactation period. Dilated pupils did not react to light. Cessation of lactation brought about restoration of vision and disappearance of fat.

Old time authors—Lawrence (18), Deval (18)—described cases of amaurosis during protracted lactation or cessation of secretion of milk—Himly (18).

G. Hemorrhage.

Leber (19) mentioned a case of retinal hemorrhage during normal menstruation.

Vicarious bleedings were described in absence of menstruation.

Heusinger (20) described a case of a hysterical woman who once menstruated through the angle of the eyelids, usually through the cheeks, often through the mammary papillæ, rarely through the hand, several times through the ear, sometimes through the stomach and nose, at times regularly through vagina.

Coursserant (9) described protrusion of the left eyeball and amblyopia at time of puberty. Hemorrhage in corpus vitreum. Capillary hemorrhages may appear as a result of high blood pressure. Therefore they were observed after an active hyperæmia during irregular menstruation; also in climacterics.

H. Many authors observed:

Inflammations of the lids, cornea, sclera, choroid, iris

Eruptions in the form of herpes take place in the conjunctiva sometimes during normal menstruation. Keratitis interstitialis, chorioiditis disseminata, sclero-chorioiditis posterior with glaucoma were observed in amenorrhea. Keratitis pannosa was seen at the time of puberty. Iritis, glaucoma, iridocyclitis were marked at climacteric. Thaon (11) observed primary iritis during suppression of menses. Antiluetic treatment was without result. Appearance of menstruation caused an improvement.

Affections of cornea were also observed by Daugenet (21), *Teillais* (22).

Inflammations of conjunctiva, cornea, and iris were marked during lactation.

In conclusion I shall mention H. Cohn's (23) case of normal menstruation accompanied by exophthalmus and resembling Graves' disease. Swelling of the thyroid gland and palpitation of the heart did not fail to appear. Nearly similar cases were described by Teillais and Morelli (24).

The above data lead us to conclude that ophthalmic disorders in women are often associated with affections of the generative organs, and that they are a result of either a hemorrhage or affection of the optic nerve and retina of a non-inflammatory nature or of some inflammatory process in the eye.

How did some of the older writers explain the relation between ophthalmic disorders and the sexual sphere, and how should it be explained at present?

Arlt (25) and Morgagni (26) were of the opinion that amaurosis in pregnancy is caused by the pressure of temporarily enlarged vessels upon the optic nerve. Here we assume pressure by the enlarged hypophysis upon the chiasm.

Weber (25) presumed that there must be an agent irritating the entire nervous system. We assume the toxins of unbalanced endocrinal correlation as such an agent.

Traube (25) thought that arterial pressure and hydræmia led to accumulation of fluids in the cranial cavity resulting in high intracranial pressure with consequent papillary œdema

and insufficient filling of the cerebral vessels decreasing the nutrition of the visual centers and producing blindness. We assume that high intracranial pressure and the concomitant results may be due to an enlarged hypophysis.

Fredericks (25) considered retention of the products of decomposition in the blood as the direct cause of amaurosis and uremia. Foerster (27) held that the changed composition of the blood was the cause of ophthalmic disorders. Deval (26) attributes blindness to nervous disorders. We assume unbalanced endocrinal correlation.

Leber (27) has said: Maybe there is some remote common cause for cessation or non-appearance of menses and accompanying neuritis.

Power (28) in his lecture on "The relation of ophthalmic diseases to certain normal and physiological conditions of the sexual organ," says: "The precise mode in which disturbance of the menstrual function operates in inducing ophthalmic disease is obscure. It may be by retaining in the blood, in cases of sudden suppression of the menses, and in cases of amenorrhea, more slowly induced materials that are of a poisonous nature, the action of which is indicated by the loss of bloodmaking power, and consequent imperfect performance of other functions, with spasms and pain in different regions of the body, in which the eye participates; or it may be by acting on the vessels of the eye alone through the nervous system in a reflex manner, and thus interfering with its nutrition, as we know may occur in cases of dental disease and in intestinal irritation due to worms."

As we have seen, all these ophthalmic disorders as observed by a multitude of scientists are of a very similar nature. This in itself makes us think that under all such conditions, a certain agent appears in the system which brings about the mentioned disturbances, and that that agent is the same at all times or at least of the same category. The circulatory disturbances are only contributory to this agent. As in all these conditions, disturbed endocrinal secretion can be suspected—efforts should be directed along this line. It is self-evident that this problem awaits a solution in the future.

Some of the observations point directly to changes in the hypophysis cerebri as the cause of visual abnormalities.

Hypophysial changes are the result of the diminished activity in the female generative glands. In this respect Prof. Finlay's observations are extremely interesting, (bitemporal contraction of visual field during pregnancy). Of interest is my own case of suppressed menses (temporal hemiopia monocular—then obesity set in); and Dr. Gibbon's case, where through three successive lactations the woman would become fat and muscularly weak. Observations of the visual field in dysmenorrhea are also of great interest.

These cases definitely point to the hypophysis as the cause. It is a fact that tumor of pituitary gland produces blindness by pressing upon the chiasm, and often with bitemporal hemianopsia. It is also well known that changes in the hypophysis leading to hypopituitarism cause dystrophia adiposo-genitalis.

It is possible that changes in other ductless glands would be responsible for the results as in Dr. Cohn's case where symptoms similar to Graves' disease developed with an enlargement of the thyroid gland during normal menstruation.

As I have said, accumulation of toxins in the blood, enlargement of the hypophysis, or both together, can easily give us a clue to the causes of ophthalmic disorders in women.

Not only eye disorders but also affection of the motor apparatus of the eyeball can be explained in the same manner, as toxic paralysis of the eye muscles is not uncommon.

In spite of the fact that the question discussed in this paper is not only of theoretical but also of practical interest, there is very little about it in the literature. Textbooks on eye diseases do not deal with this subject. In one of the best textbooks, that by Prof. Fuchs (edition 1921), nothing is said on this subject. Therefore, it seems to me timely to discuss this question in the press and at meetings.

CONCLUSIONS.

1. Ophthalmic disorders of genital origin in women may depend upon unbalanced correlation between the glands of internal secretion.

2. As a result of this unbalanced correlation, an accum-

mulation of toxins in the blood takes place and often enlargement of hypophysis cerebri.

3. Ophthalmic disorders may be a result of pressure of an enlarged hypophysis upon the chiasm, or action of the above mentioned toxins or both simultaneously.

4. In all cases of irregular menstruation or in the presence of other symptoms of unbalanced correlation, systematic examinations of the visual field and fundus are desirable.

5. In experiments on animals, attention should be paid to the changes in the eyes and to the nature of visual disturbances when they occur.

6. It is important to investigate the action of various glandular extracts upon the ophthalmic disorders resulting from faulty correlation, and

7. Investigate the action of ovarian transplantation upon same.

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A CASE OF NEUROFIBROMATOSIS WITH BUPHTHALMUS.[1]

By Dr. ALFRED WIENER, New York.

(*With four illustrations on Text-Plate XVII.*)

NEUROFIBROMATOSIS, or rather von Recklinghausen's disease, is a rare and interesting condition, first described in 1882.

Osler tells us there are four essential features which characterize this malady.

First: Soft fibrous nodules varying in size and number scattered over the surface of the body.

Second: Tumors resembling plexiform neuromata present on any part of the nerve trunks from their central origin to the periphery.

Third: The presence of patches of brownish pigment of the skin, either as small spots or large areas.

Fourth: Many variable sensory or motor phenomena resulting from the presence of nerve tumors.

A disease which Fuchs says is compounded of a number of changes, mostly congenital; such as fibromata, plexiform neuromata, flat pigment moles, lymphangiomata of the lids and orbit, unilateral hypertrophy of the face, with involvement of the terminal filaments or end organs of the nerves, and other times of the larger nerve trunks themselves.

At times it may only affect the fifth nerve.

The occurrence of a buphthalmus in connection with a neurofibromatosis of the fifth nerve, in which there was an involvement of the lids, jaw, and forehead on one side of the

[1] Presented at the meeting of the Section on Ophthalmology of the New York Academy of Medicine, January, 1924.

head, was recently observed by me in the ophthalmological department of St. Luke's Hospital.

The patient was a male child two years of age. The father and mother were perfectly healthy, with no hereditary history of any kind. The child directly after birth appeared to the mother as having a very prominent eye. Gradually this became more noticeable, and the face at the same time became distorted. The whole left side seemed to be larger than the right. The lids appeared thick and the forehead seemed to project forward. (Fig. 1.)

The parents' natural anxiety led them to consult various clinics, with the natural disappointing result that no relief was obtainable. As soon as the rarity of the condition was recognized, the patient was admitted to my wards at St. Luke's Hospital, and a painstaking physical examination was made. The child was a well nourished and remarkably strong and healthy looking baby. On its body were noticed two fair sized pigment patches, one just above the clavicle and the other just above the groin. What was especially noticeable about the child, was the disfigurement over the entire left side of the face. This whole side was more prominent than the right. The cheek looked swollen and the upper lid was thickened. On opening the mouth, it was noticed that the left jaw was distinctly larger than its fellow on the opposite side, and contained a number of erupting teeth. The left eye was apparently blind and the typical picture of a buphthalmus. Over the forehead just above the brow, a mass could be felt underneath the skin, which gave one the sensation of a bunch of worms. The whole orbit was larger in circumference than the right. This was all the more evident after an X-ray examination had been made. This picture showed the left orbit when compared to the right as increased in all of its dimensions. The left malar bone was much more dense and larger than its fellow on the right side. (Fig. 2.) The tongue and soft palate were normal. The interior of the nose showed no abnormalities. A chemical and microscopic examination of the blood was negative. A Wassermann was also negative. A subcutaneous test with tuberculin showed no evidence of a reaction either local, focal, or general. Heart, lungs, and kidneys showed no anomalous condition. A lumbar puncture was also negative. An X-ray of the skull showed the sinuses clean and the sella turcica normal. With the exception of the fifth nerve, all the cranial nerves were normal to their respective reactions, as far as I was able to determine in a child of this age. The optic nerve could not be seen.

ILLUSTRATING DR. ALFRED WIENER'S ARTICLE, "A CASE OF NEUROFIBROMATOSIS WITH BUPHTHALMUS."

FIG. 1. Photograph of child showing the hemihypertrophy of the left side of the face. The X marks the location of the neurofibroma beneath the skin.

FIG. 2. X-Ray photograph of the skull showing the enlarged orbit on the left side and the increased density of the malar bone.

FIG. 3. The neurofibroma removed from underneath the skin of the forehead.

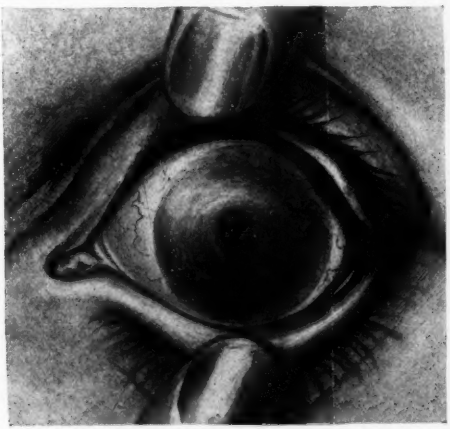

FIG. 4. Case of neurofibromatosis with buphthalmus. Represents the network of grayish fibers seen in the cornea, probably hypertrophied nerve fibers.

Examination of the eye: The cornea is very much enlarged in all its dimensions. In width it measures 16*mm.* There is to be seen, apparently in its superficial layers, a network of straight and curved lines which appear to be lost just beyond the corneoscleral margin (Fig. 3). The anterior chamber is deep. The lens was not dislocated but very hazy. The iris looked thin and atrophic and was widely dilated. The fundus was invisible. The movements of the eye were normal when following the other eye. The left eye alone could not be made to follow a light. To all tests it was practically blind. Tension taken with a Schiotz tonometer at various times was always within the physiological limit.

On account of the marked disfigurement, the eye was enucleated. At the same time a portion of the mass beneath the skin of the forehead was also removed. (Fig. 3.)

Microscopic examination of the eye: The parenchyma showed no abnormality. The surface epithelium, Bowman's membrane, and the Descemet membrane showed no break or pathological change. Although an endeavor was made to demonstrate the presence of thickened nerve fibers, in the cornea, we were not successful, on account of apparent faulty staining. I am therefore unable to say just what the visible network of fibers seen in the cornea really are. (Fig. 4.) A number of elongated cells were found in the sclera close to the substantia propria of the cornea, which may be thickened nerve fibers.

The anterior chamber is deep. On the nasal side there is an absence of the ligamentum pectinatum and Schlemm's canal. On the temporal side there is a similar absence and the angle is filled with what I should judge to be persistent fetal tissue. The iris looks atrophied but otherwise there is nothing pathological about it. The ciliary body is flattened but its blood vessels are well filled. There is no neurofibromatosis of the iris or ciliary body. There was nothing of importance in the sclera, with the exception of the vessels which were distended with blood, especially the veins.

The choroid occupying the posterior portion of the globe is thickened due to an abnormal overgrowth of fibrous tissue, highly nucleated and rich in pigment cells. The vessels are filled to their full capacity. Scattered throughout the choroid are a number of small light-colored oval bodies. These are probably hypertrophied nerve end organs. Under high power these structures appear distinctly laminated. In the anterior portion of the globe the choroid approaches the normal, but in some places appears thinned. Most of the ciliary nerves, where they have been

met with, either on longitudinal or cross section have been found affected. There was present a general hyperplasia of the fibrous tissue. Both the endoneurium and perineurium were thickened. The retina appeared normal with the exception that in several places there were present surface hemorrhages. The optic nerve showed a distinct cup with atrophy of the nerve fibers.

The mass removed from the forehead consisted entirely of nerves and fibrous tissue. The peri- and endoneurium were decidedly thickened. The whole picture was characteristic of neurofibromatosis.

The findings in this case coincide very much with the findings reported by other authors. One of the first to call attention to the association of neurofibromatosis with buphthalmus was Adolph Sachsalber. He reported the case of a child with left-sided hypertrophy of the face with buphthalmus. He explained the diseased condition as due to a disturbance of the trophic fibers of the nerves of the choroid, with a secondary disturbance in this membrane. This led to a diffuse overgrowth of connective tissue, and a diseased condition of the perichoroidal lymph spaces and lymph passages along the vortex veins. Through this lymph circulatory disturbance there was an impeded outflow and the buphthalmus occurred.

Lezius next published a case of left-sided buphthalmus with left-sided hypertrophy of the lids. He was of the opinion that a common disturbance of development was the origin of the trouble. This might express itself in the eye as hydrophthalmus, and in the skin as a simple hypertrophy.

The next important cases were reported by Snell and Treacher Collins. They reported as findings tissue rich in nerve fibers, with endo- and perineurium thickened. The globe showed thickened scleral nerves and thickening of the nerves of the cornea. There was an attachment of the root of the iris and an absence of the ligamentum pectinatum. Collins in an article in which he notes the pathology of two eyes, discusses the question whether the enlargement of the globe is in some way directly connected with the neurofibromatosis, or whether it is brought about, as is usual in most cases of buphthalmus, viz., by an increase of tension, due to some

maldevelopment of the channels of exit for the intraocular fluid. In the eyes examined by himself, he found considerable malformation about the angle of the anterior chamber. The spaces of Fontana did not seem to have been developed. In each eye there was a broad adhesion of the iris to the posterior surface of the cornea, at its periphery, these structures never having become separated during fetal life. He thinks that although the buphthalmus might be accounted for in this way, there is every evidence that individual parts of the eye do become affected by this condition of neurofibromatosis. From this study of Collins one can understand that all portions of the ciliary nerves may be affected. That in the uveal tract, as in the skin, there may be a general hyperplasia of the fibrous tissue. In some cases only the terminal filaments or end organs of the nerves are involved, and in other cases the larger nerve trunks. The reason that the retina is never the seat of this affection, is probably because it is entirely derived with the exception of its blood vessels from the neural epiblast. Neurofibromatosis is essentially a hyperplasia of tissue of mesoblastic origin. A little later Siegrist published a case of congenital buphthalmus and elephantiasis of the left upper lid, cheek, and temporal region. He was also of the opinion that the disease of the ciliary nerves and the buphthalmus were in a certain way associated.

The next year at the Heidelberg Congress, this case was again brought up for discussion by Rabinowitsch. She considered the buphthalmus as due to a paralysis of the vasomotor branches of the ciliary nerves, producing a general hyperemia of all the membranes of the eye. As a result of this, an increased secretion developed. This chronic augmented outflow through the ligamentum pectinatum and Schlemm's canal, with a probable changed and altered lymph, produced an adhesion and thickening at the angle of the anterior chamber, and as a result an increased intraocular tension. Cabanes who reported a case of buphthalmus of the congenital type with striking hypertrophy of the face, came to the conclusion, that the enlargement of the eye was dependent upon a condition of hypernutrition. In this case there was a large deep seated angioma in the temporal region. There was no obstruction in the excretory passages. In

Sutherland's and Mayou's case cited in Elliot's book on *Glaucoma*, as an example of Recklinghausen's disease, the right side of the head and face was enlarged; the branches of the third division of the fifth nerve could be felt as so many cords, and the lids were hypertrophied. White streaks in the cornea were taken to represent enlarged ciliary nerves. The tension was up and the disk cupped. Reis expresses the opinion, that where a hemihypertrophy of the skin occurs in connection with a congenital buphthalmus, this association is the result of a spreading of the disease to the eye, beginning in the fetal period and later localizing itself in the neighborhood of the eye. He believes the buphthalmus a secondary process due to tension disturbances. At the 35th Heidelberg Congress Michel expressed himself in regard to the association of neurofibromatosis and buphthalmus as follows:

First.—That the simultaneous occurrence of both forms is not accidental.

Second.—In view of the fact that the location and grade, as well as the distribution, of the one-sided hypertrophy of the face may vary, a buphthalmus may or may not be present.

Third.—That the one-sided buphthalmus can in the absence of other stigmata be the only sign present of a one-sided facial hypertrophy.

He believes the fibromatosis of the ciliary nerves and the buphthalmus are the result of a coördinated disturbance. The production of the buphthalmus he attributes to the result of a primary fibromatosis of the angle of the anterior chamber, brought about by a disturbance of the mesenchyma in the early fetal period. Seefelder disputes this last theory, having examined very carefully an eye of this type, and found microscopically no fibromatosis present. He found in the angle of the anterior chamber some changes which evidently produced filtration disturbances. These changes were in the nature of persistent fetal tissue present normally in the eye during the fetal period.

The microscopical findings in the case reported by Komoto and Murakami agree in general with the findings of Sachsalber, Snell, Treacher Collins, Rabinowitsch, and Michel.

When it comes to the argument as to what relationship

exists between buphthalmus and neurofibromatosis, which has been answered so differently by various authors, according to their pathological findings, Komoto and Murakami believe that the difference of opinion is due to the fact that most of the eyes have been examined at different stages of the disease. They believe that during the long period of the disease, much can through the increased tension disappear, and much can arise anew, so that it would be difficult to say what is primary and what is secondary. In their own case, they believe that due to a disturbance of the circulation an increased secretion occurred, which gave rise to an increase of the tension. They believe it possible that hypersecretion can be brought about through circulatory disturbances. This occurs indirectly through the enormous development of connective tissue, which not only affects the ciliary nerves but also the blood-vessels and the lymph vessels to such an extent that a stasis results. The closure of the angle and the obliteration of Schlemm's canal they consider a secondary matter. It is hard for them to believe that a paralysis of the blood vessels, brought about by an alteration of the ciliary nerves, would result in increased tension. Experimental work has shown that under such conditions rather a hypotony would result.

In all these cases which have been reported including my own, three conditions stand as predominating.

First.—An actual hyperplasia of tissue.

Second.—An actual hyperemia.

Third.—A distinct thickening of the nerve fibers at some portion of their course.

In my own case, we have an hypertrophied malar bone, an hypertrophied lid, a neurofibroma underneath the skin of the forehead with some thickening of the skin, and an enlarged eyeball lying within an enlarged orbit. Clinically the eyeball was recognized as a typical case of buphthalmus. At no time could I detect any increase of tension, and such tension was tested with the Schiotz tonometer a number of different times.

Might it not be possible that the enlarged eye as well as the orbit are simple cases of hyperplasia coming under and being brought about through the same influences which have produced the hypertrophy of the lid and malar bone? Again

is there a possibility that two factors were at work to produce the buphthalmus in this case? One the fibrosis and the other a periodic increase of tension due to circulatory and lymph disturbances. I confess that until the ultimate cause which determines the growth and decrease of tissue is known, not much in the way of an explanation can be offered for the above symptom complex. At present it seems to be beyond the reach of scientific investigation.

A knowledge of the disease ensures an easy diagnosis. The prognosis should be guarded. While the disease may remain stationary for an indefinite period, and be compatible with fair bodily health, the probabilities are that sooner or later it will progress, become general, and prove fatal A sarcomatous transformation of some of these overgrowths is not unknown. I wish here to express my thanks to Dr. Leila C. Knox for her kind assistance in the examination and preparation of the pathological material.

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REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. DAVID H. WEBSTER, Secretary.

MEETING OF MARCH 19, 1925. DR. CONRAD BERENS, CHAIRMAN.

Dr. Ross Hall Skillern of Philadelphia, read (by invitation) a paper on **headaches of the nasal aspect.** The author called attention to the pathological factors entering into the causation of headache. Pain, both as to the character, degree, and even time of appearance, depends largely upon the pathological condition present within the sinus, the most important being some form of pressure. This pressure may result in two ways—one in which the mucosa becomes so swollen as to impinge on the mucosa of the opposite side, the other by occluding drainage and a third form which the author seldom finds, is negative pressure, or so-called vacuum headache. Little reliance can be placed upon the diagnostic value of the periodicity of the typical sinus headache. Active inflammations usually produce a sickening throbbing symptom, while chronic condition cause a dull ache. Another character of sinus headache is its peculiar intolerance to alcohol or tobacco. The subjective localization of the pain is important for while a certain sinus is involved the pain will always be located in the same place. Each of the sinuses was then taken up individually, and the location, duration and character of cephalgia discussed.

Dr. George E. deSchweinitz of Philadelphia, read (by invitation) a paper on **ocular headaches.** After a brief reference to the history of asthenopia, ocular headaches and eyestrain reflexes, with special reference to the pioneer work of Donders, and of Weir Mitchell, William Thomson and Ezra

Dyer in this country, followed by a discussion of the frequency, etiology, varieties, development and associations of eye headache, Dr. deSchweinitz summarized his observations as follows:

Eye-strain due to refractive error, heterophoria and accommodative dysfunction, is responsible, in whole or in part, for about 60 per cent of functional headaches; small error of refraction are often more potent in this respect than the larger ones; all types of refractive error and muscle imbalance are capable of causing headache, simple and compound astigmatism and hyperphoria being the most potent refractive defects in this list; the eyes themselves may be free from pain, and the headache none the less be of ocular origin; physical vigor does not necessarily exempt patients from ocular headaches, although often such headaches do not appear until physical power fails; there are no pathognomic features of eye headache, which may, however, be strongly suggestive, they may be situated in any portion of the cranium, and be of any degree of intensity, and may closely resemble those usually ascribed to other causes; the same patient may have several kinds of headaches, which differ in their manifestations, one variety being due to eye-strain, and others to different causes; eye headache may immediately follow the visual strain, or be apparent only long after the eye work is completed; ocular work at close range is not a *sine qua non* of the head-pain development; long distance ocular effort may be equally effective; ocular headaches may be acute, sometimes chronic, but chronicity is usually suggestive of some additional factor directly contributory, or they may be recurrent or periodic; eye-headache appears at times to exercise a certain quality of selection with respect to the "agent provocateur," for example, writing may be pursued with impunity, but reading quickly produces head-pain, moreover in exactly the same circumstances.

Dr. de Schweinitz summarized the long list of the so-called eye-strain reflexes and the many nervous and other phenomena which refractive errors and heterophoria originate in addition to headaches, and emphasized their widespread evil influence. He urged the most painstaking care in the measurement and adjustment of correcting lenses, and called attention to the need of coöperation between general physicians and ophthalmogists and other doctors trained along special lines in the

management of the complicated cases, in order that all factors may be disclosed and the best therapeutic results may be achieved.

Dr. LUDWIG KAST read a paper on **headache from the viewpoint of the internist.** Until the different mechanism producing headaches are sufficiently known it will be impossible to give an etiological or pathological classification of them. It is therefore still necessary to resort mainly to clinical classifications to bring some order into the great variety of headaches.

Aside from the traumatic and symptomatic headaches all forms may be divided into the extracranial and intracranial, though many intracranial headaches are due to reflexes from extracranial sources. Among the intracranial headaches the most interesting problems concerns migraine which in the light of recent studies appears to be a syndrome due either to such definite disturbances as cerebral arterio-sclerosis, brain tumors, sphenoid infections and others which may be termed symptomatic migraine or due to a disturbance, the mechanism of which is still unknown, and for which a variety of different hypotheses exists. This form may be termed essential migraine. The latter form can be best explained by disturbance of the pituitary gland. Such disturbances could occur, even though there are no structural changes in the gland by paroxysmal swelling of the gland similar to the transitory enlargements occurring in other endocrine glands such as the ovaries, thyroid, etc. The reason why in some individuals such enlargement of the gland will cause migraine and in others will not, depends upon some anatomical peculiarity which will cause pressure and malfunction due to relational anatomy of the pituitary gland and adjacent tissues.

Based upon the anatomical studies of Dr. J. Parson Schaeffer, Professor of Anatomy at Jefferson Medical College on 1925 freshly exposed brains and based upon clinical studies, Professor George E. deSchweinitz pointed out how pressure symptoms upon the optic nerves and the optic chiasms as well as upon the arterial circle of Willis will come about if anatomical conditions are favorable to pressure symptoms.

According to Dr. Kast in studies on more than 100 cases of migraine, the majority of cases show radiologically either a small or a partially enclosed sella turcica. From Schaeffer's

anatomical studies it is evident that the roof over the sella turcica in its slanting, density and opening for the infundibulum would determine whether pressure symptoms would arise if the gland becomes enlarged even though the sella turcica may not radiologically appear abnormal.

Undoubtedly aside from the mechanical disturbances abnormalities of the pituitary secretion may account for some of the symptoms such as nausea, vomiting, polyuria, vasomotor disturbances, etc.

The pituitary hypothesis of migrane, as suggested by Deyl twenty-five years ago would therefore present migraine as pituitary paroxsyms of which the headache is one of the outward manifestations. This might explain why almost all cases of migraine have tendencies to other endocrine disturbances, and it also explains why migraine attacks are preceded before puberty by spells which show all earmarks of endocrine disturbances without headaches. After climacterium the attacks of migraine become less frequent or disappear or are replaced by peculiar spells of depression, indigestion, malaise without headaches or with very little headaches.

The therapy of headaches as heretofore has been merely symptomatic, but small doses of pituitary in the interval, ovarian extract for the week preceding menstruation, iodine and small doses of thyroid in hypothyroid cases with migraine have shown better results than any other form of symptomatic therapy.

The neurological aspect of headache was presented by Dr. FOSTER KENNEDY.

DISCUSSION: Dr. EDGAR S. THOMPSON: Dr. de Schweinitz's most interesting paper deals most concisely with the broad aspects of the subject and is so sane and well balanced that it is not possible to differ with the statement he makes. I can only, therefore, particularize in what seems to me some of the most important features. My own feeling is that astigmatism is by far the most common cause of ocular headaches. It is undoubtedly due to cramp of the ciliary muscle in greater or less degree, in more acute or chronic types depending on the proportion of the causative factors, the outline of which he has so well given. The exact mechanism of the muscular action is still a disputed point. Meridional contraction, lens

tilting, and interpretation of diffusion circles, have all been cited as causes for the efforts at compensation or what we might call nature's effort at correction of the mechanical error, which in turn causes the cramp. I am rather a firm believer in the theory of meridional contraction, or contraction of a definite meridian of the circular ciliary muscle. It is difficult otherwise to explain those cases in which the cornea shows two or even three diopters of astigmatism and yet the vision is normal. The researches of Hansen and Voelckers, as long ago as 1868, seem to show that such contraction can occur. Probably an over development of certain fibers takes place which permanently alters the shape of the lens, though on the other point anatomical evidence is lacking. As the ciliary muscle normally contracts completely it follows that an abnormal action of the muscle would be much more apt to produce cramp, just as a twisted arm would be more apt to have muscular cramp, than one which acted naturally. Low degrees of error, which can be compensated, are perhaps more apt to give headache or other reflex symptoms, than the higher degree cases. The higher degrees suffer from diminution of vision. Dr. de Schweinitz has emphasized this point. Another important point in the paper is that muscular tone does not exempt an individual from astigmatic strain. Any ciliary muscle however strong can become cramped if the astigmatic habit continues long enough. The diagnosis then can only be determined by careful examination of the eyes, and yet I know many general practitioners who have a very marked ability to sense ocular headaches and whose cases, when sent to me, almost always require glasses. I therefore think that the eye-strain headache has features which are often quite suggestive if the clinical characters are carefully observed. With regard to the headaches caused by muscular imbalance, the problem is more difficult. Such headaches, particularly those caused by vertical deviation of the eyes, are more apt to be occipital; are accompanied by vertigo, or other severe symptoms of nerve irritation, even as severe as convulsive seizures. In such cases much depends on the vulnerability of the nervous system in question. It is illogical to suspect these unusual reflexes in individuals of good tone. Such individuals are more apt to have direct eye fatigue, from astigmatism or

from muscular errors, and secondary headache. The tendency to ocular headaches changes markedly with age, and is at its worst in the young and in early adult life, as a rule. After the presbyopic period sets in ocular headaches are apt to be less severe and local fatigue becomes a more common symptom. Persons whose reading glasses are not up to the mark simply do not see. In elderly people, headaches of a certain type confined to the temple, are sometimes due to border line types of glaucoma, in which the ocular tension rises to thirty or thirty-five millimeters, and yet the eye does not become red. I only mention this in passing as the paper does not discuss this part of the subject. Finally I wish to heartily endorse Dr. de Schweinitz's final paragraph. We have gotten very far away from the conception of the eye as a mechanical instrument almost independent of the physiological processes of the body. When it comes to securing the maximum efficiency, or even bare comfort in some cases, it is necessary to work closely with the general practitioner, and to take into consideration the problems of digestion, circulation, and innervation, in fact everything which pertains to the highest degree of functional activity, a rather large field.

MEETING OF APRIL 20, 1925. DR. CONRAD BERENS, CHAIRMAN.

Dr. MARTIN COHEN presented a case of **asteroid hyalitis** in a man aged 60 years, physical examination normal. Vision in both eyes $\frac{20}{20}$ with correction. External examination, intraocular tension and the pupillary reaction were normal. The fundus examination of both eyes was also normal of angiosclerosis.

In the vitreous of the left eye one sees mainly whitish globular masses, about the size of the head of a pin, and a few angular shaped ones. In the periphery is seen a collection of these opacities in masses resembling the milky way. The spherical bodies are suspended in the vitreous mesh work at various levels. They move slowly when the patient is directed to move the eye in various positions. This condition as a clinical entity was first described by Benson. Four years ago Vierhoff published a microscopical and microchemical study of this pathological condition.

Lantern slide demonstration was presented, first of a case of

sub-conjunctival luxation of lens, occurring in a colored man 40 years old. Patient received a blow on the left eye with a bootjack. A few hours after the injury the eye was examined. There was swelling of the lid with sub-conjunctival hemorrhage; the tension was reduced. There was a globular mass situated at the sclero-corneal junction covered by the conjunctiva. The eye was painful to palpation. Hemorrhage filled anterior chamber; there was no light perception present. The eye symptoms became more evident and pain increased. Enucleation advised, but was temporarily refused. Three weeks after the injury enucleation was again advised and accepted by the patient. The eye was removed and placed in 5% formalin for future study.

Some seventy-five eye sections were made and carefully studied. There was no evidence of rupture of the conjunctiva in any of the sections. The lantern slide demonstration showed hemorrhage in anterior chamber and vitreous. The anterior segment of the eye showed a rupture of the sclera near to, and connecting with the corneo-scleral junction, associated with rupture of the iris root, producing irido-dialysis. At the time of the blow the zonula fibres on the trauma side ruptured leaving the iris and ciliary processes torn away by the adherent zonula fibres; they are seen in the rupture. The pupil was displaced toward the side where the iris was intact. Lens was seen under the conjunctiva. Exudate is seen beneath the choroid showing phagocytic leucocytes carrying pigment. Foci of lymphocytes are seen in the choroid suggesting beginning sympathetic ophthalmia. There is detachment of retina with blood clot beneath it; papillary œdema with deposit of pigment-bearing-phagocytes. These wandering phagocytes are also seen in the nerve stem.

Second case of lantern slide demonstration: Specimen taken from an eye enucleated from a man 30 years of age, who sustained an injury eleven years previously, producing **phthisis bulbi.** The eye was small and misshapen without any light perception. The cornea showed a scar and traumatic cataract with synchysis scintillans in the anterior chamber. The eye after enucleation was fixed in gelatin and frozen sections were made. Some of the sections were stained with Sudan III. These sections showed cholesterin crystals in the anterior

chamber of the vitreous, also in the exudates. Photographs were taken of the frozen sections showing the double refracting fats in the chambers of the eye, through the polariscope.

DISCUSSION: Dr. LINN EMERSON said fifteen years ago a man came in to see him, who was struck in the eye with a shovel, causing a dislocation of the lens under the conjunctiva, down and out. Dr. Emerson ordered him to go to the hospital. He did not go. Dr. Emerson did not see him again until five years later, when he came to his office, with his eye perfectly quiet, and his vision $\frac{20}{100}$. He had treated himself at home with tea leaves.

Dr. VANDEGRIFT demonstrated a **rotary bifocal,** with a spring attachment making it possible for the wearer to place the segment in the upper field of vision by a simple twist of the lens.

DISCUSSION: Dr. LINN EMERSON thought that his trifocal glasses were more scientific. Some time ago he had attempted to have a pair of trifocals made up, and on top of that a 45 which they said could be made at a great expense. Four months ago Bugbee came out with the same glass. It consists of an arbitrarily plus 1.37 for intermediate, which gives a range from 14 to 29 inches, and an additional reading below. Dr. Emerson thought that for the nose and throat doctor, they were a very valuable asset.

Dr. HERBERT W. WOOTTON read a paper on **heterophoria** in **ametropia, non-surgical treatment.** Published in this number.

Dr. J. W. WHITE spoke on the **surgical treatment of heterophoria.**

Dr. P. CHALMERS JAMESON read (By invitation) a paper on **principle of muscle recession** with scleral suturing, as supplied in the correction of heterophoria and squint. The author of the paper reviewed the origin of the recession in 1922 and made a comparison between the manner in which advancement and tenotomies were performed, the precision, gradation and definite anchorage to the globe in the former and the lack of these procedures in the latter.

The adoption of these principles as applied in advancement to tenotomy, almost completely revolutionizes the operation of simple tenotomy as to the amount of correction obtained and the protection to convergence secured.

A definition of the term recession now beginning to be frequently heard in place of tenotomy was "the backward replantation of a muscle and capsule flap in bulk, graded as to amount of retroplantation and held in position by direct scleral suture at or near the equator."

The principles were discussed under different headings conforming to the steps of the operation.

The conjunctival incision should be made at or about the curve of the semilunar fold, pulling the loose wrinkles of conjunctiva from beneath, leaving an ample flap of conjunctiva on the corneal side of the incision. If this is not done, the conjunctival wound will not close as the portion on the corneal side will not stretch to point of recession or fixation of muscle end to the globe.

The value of thorough exposure of capsular and muscle field was most necessary and the knowledge obtained of capsular and muscle conditions existing, of great value. The taut capsule, a condition frequently recognized, could be incised equatorially above and below almost to the border of superior and inferior recti when this condition obstructed free excursion. This undertaking could be done with impunity if scleral fixation was practiced.

Marked emphasis was placed on the necessity of retroplanting the capsular and muscle flap in bulk. The muscle should never be stripped to its bare fiber elements and the adipose and connective tissue surroundings of the large flap of muscle and capsule should not be mutilated. Especially is this to be respected with the profuse areolar tissue between the surface of muscle and caruncle.

The object of the recession operation was not to weaken the muscle but to protect convergence after the extended retroplantation was performed by leaving the muscle strong and unimpaired.

The technique of the non-perforating suture was described. Practice first on the enucleated eye, was advocated. A large blunt needle was useless. It would burst and mutilate fibers. A slender full curved sharp needle 10*mm* from tip to eye, was best. The moment the point of the needle becomes indistinct it is made to approach the surface. The needle, after counter puncture, should be visible through the translucent sclera

through its entire continuity as also the suture after it is drawn.

In the matter of gradation, conservatism was advocated. A simple rule was 5*mm* recession in the higher deviation, 2½ to 3*mm* in the lesser. A careful computation based upon the size, breadth, laxity or tension of the muscle and nature of the squint, was advisable. A striking and gratifying phenomena was the ability of the muscle to adjust to convergence required.

The author of the paper gave a brief discription of the operation which was to be found in the ARCHIVES OF OPHTHALMOLOGY, 1922. He commented upon certain modifications which had been suggested and advocated, and believed the single suture operations were not sound, as they made for a pointed attachment. All these should be supplemented with wing sutures as one of the principles of recession is a broad attachment of the muscle to the globe.

The operation recently advocated in New York which anchored the muscle to the sclera and then tying to muscle stump was one which he had discarded earlier in the experimental work, as it did not directly suture to the sclera. If practiced, it should be supplemented with wing sutures.

In conclusion the writer stated that in every operation, advancement or recession, there would be found post-operative conditions, neurological, muscular and connective tissue obstructions which are the contributory causes to failures or partial failures in well regulated operations and gave an erratic quality to final results, and recessions were no exceptions. He was sure, however, that no single operative procedure would yield as large or as staple or uniform correction in strabismus, than recession in bulk with scleral fixation by scleral suturing.

DISCUSSION: Dr. ELLICE M. ALGER said: I find myself in very complete accord with all the papers. There are one or two points which I should like to emphasize. Dr. Wootton has stated that most esophorias, for instance, are due to uncorrected hyperopia and if taken early enough yield to convex glasses. Exactly the same kind of strain is imposed on the eyes in young children by the almost universal childhood habit of holding a book six inches from the eyes instead of sixteen, of lying on the floor or face down on a bed to read. It causes spasm of accommodation and co-incidently, of conver-

gence as well—it is the great factor in the pseudo myopia in school children and finally forces the predisposed over into actual progressive myopia. The same process occurs in the late thirties in what we sometimes call subnormal accommodation, as well as in the early presbyopic period, where the over-stimulation of one tired muscle results in the over-stimulation of other synergistic ones.

We all have "phorias" of one sort or another which are kept from becoming "tropias" by aid of our fusion powers; which means controlling one set of over-stimulated muscles by over-stimulating their antagonists. The waste of nervous energy is obvious, as well as the possibility of discomfort and fatigue. If we can so arrange matters that this "compensation" is done with relative ease we have solved our problem. There are many cases, however, where by reason of anisometropia—muscular paralyses and the like, fusion is likely to be very difficult and where it would be greatly to the patient's advantage, to frankly abandon it. I have made a good many patients perfectly comfortable for instance by correcting one eye for distant vision and the other for near, though in many cases it will not work at all. Or we can make them use one eye for everything just like a strabismis patient. You may remember hearing Professor Gullstrand tell how convenient he found it to be able to disassociate the eyes and wear a different focus on each. It seems to me we have almost made a fetish of binocular vision for fear of amblyopia exanopsia, concerning the existence of which I am very skeptical. In the same way I am sure we often exaggerate the necessity of binocular fusion in practical depth perception. Many strabismics or patients with only one first-class eye play good tennis and golf which are supposed to require stereoscopic vision. Statistics on the eyesight and muscle balance of our athletes, particularly ball players would be extremely interesting and valuable. I have often thought there was actually a heterophoric type of patient—always tense even to the point of irritability—always meeting things more than half way, rarely relaxed and composed—and I am sure that conditions of depressed health or over fatigue create temporarily both the nervous insufficiency and irritability which are at the bottom of the heterophorias we are discussing.

In the treatment I would therefore stress a search for possible underlying health defects and particularly for the habits of work which occasion ocular strain. But the great thing is rest and relaxation, to be secured by full correction of hyperopia and astigmatism and very likely extra help at the near-point, perhaps for the time being all the eyes will accept. Prisms, I rarely prescribe, except in hyperphoria. Exercises have their place of course but only when muscles and nerves are rested. Stimulation of tired muscles and nerves can only be harmful in the end. Much of the benefit attributed to prism exercises results from the patient's sudden discovery of the knack of coördinating muscle actions, which is of course valuable if not misinterpreted. So far as surgery is concerned, I agree with Dr. White, that it is a last resort and that the operation depends entirely on the analysis of the case before one. But I could almost summarize my position in the statement that in most cases "if you could take care of the refraction with all that this implies, the 'phoria' will take care of itself."

Dr. DUANE: In the treatment of Heterophoria we must seek to eliminate one after another the possible causes of the condition or its symptoms. The first thing to do is to correct the refraction. This alone may eliminate the symptoms so that no further treatment is needed. The correction of the refraction has also a direct relation to the heterophoria itself, as for example in the convergence insufficiency occurring in presbyopes, in whom the giving of too strong a reading glass causes trouble by imposing undue strain on the weak convergence. Failing relief by glasses, we may apply prisms for wear. I use these rarely except in heterophoria. Next we may employ exercises with prisms, etc., these being serviceable mainly in convergence insufficiency. Then we should eliminate conditions of irritation in the eye itself (conjunctivitis being common in these cases), and relieve causes of direct strain (improper lighting and wrong methods of work). If the condition persists we must seek the underlying causes often producing convergence insufficiency and sometimes other conditions. Such causes are nasal disease with or without sinus trouble, gastro intestinal disorder, tonsillar or dental infection and particularly disturbance of circulation which seem very often

to be frequent. Last of all we resort to operation. In this I should in general follow the lines laid down by Dr. White. But every case requires individual consideration. Dr. Jameson's recession seems to me to offer a very distinct addition to our armentarium, especially in the treatment of convergence excess, in which it relaxes the tension without impairing the converging power.

REPORT OF THE TRANSACTIONS OF THE SECTION ON OPHTHALMOLOGY, ROYAL SOCIETY OF MEDICINE.

(Meeting with the Comparative Medicine Section)

By Mr. H. DICKINSON, London.

FRIDAY, MAY 8TH, 1925. SIR ARNOLD LAWSON, K.B.E., PRESIDENT OF THE SECTION OF OPHTHALMOLOGY, IN THE CHAIR.

Cases.

Mr. A. Hugh Thompson showed a case of **"snowball opacities"** in the vitreous of one eye. The patient was a man aged 62, and his right vitreous was filled with round white bodies, which moved in all directions with every movement of the eyeball. The fundus appearances were normal, and the condition did not appear to be detrimental to the vision: this was $\frac{6}{12}$ in the right eye, $\frac{6}{18}$ in the left. The condition was referred to by de Schweinitz, who alluded to two American authors with whom originated the term "snowball opacities." The little bodies were larger and duller than cholesterine crystals. He had not previously seen a similar condition. Some had suggested it might be a congenital matter, but this man did not notice anything amiss until late years.

Mr. T. Harrison Butler said that in a case of his with a similar appearance the man had since developed a very resistant glaucoma.

Mr. Hamilton Kirk exhibited **a cat with tubercular keratitis** in an early stage. He said the disease was frequently met with in these animals, and he expected this cat to develop

other lesions later, such as retinal detachment. This cat could be felt to have some irregularities on the kidneys, probably tuberculous. He said that ocular lesions were frequent in distemper in both dogs and cats; 60% of dogs with distemper seemed to have eye troubles. The nearest human disease to distemper was measles, and in this latter eye conditions were not infrequent. In dogs the eye affection seemed to be due to spread of infection through the nasal duct, but this would not explain it in those cases of distemper, in the early stages, in which there was no nasal discharge. The first indication often was a milky opacity of the cornea. This might pass away, or it might persist for weeks and result in ulcerative keratitis. There was, he said, a catarrhal form of distemper in cats, with much chemosis and protrusion of swollen conjunctiva over everted lids. The animal had photophobia and suffered much pain, and a creamy material glued the lids together.

Mr. HUMPHRY NEAME referred to a paper by the late Mr. Coats describing 8 cases of tuberculosis in cats, in all of which both eyes were affected. In those there were masses of tuberculosis in the choroid, and various degrees of retinal detachment.

Eye Diseases Common to Man and Animals.

The remainder of the evening was devoted to a discussion on this subject. Mr. HUMPHRY NEAME opened with a paper entitled "Parenchymatous Keratitis in Animals," and it took the form of an epidiascopic demonstration of slides of cases of various kinds.

The first case was that of an animal whose eye was ruptured by a turnip being thrown at it. In five days a very acute affection of the eye had taken place, and clouds of nuclei could be seen between the lamellæ. The anterior chamber, between the cornea and iris, was occupied by purulent material, and there was a layer of pus between the ciliary body, the iris and the lens. There was active suppuration, and large numbers of polymorphonuclear leucocytes were evident in the section. The greatest area of infiltration was a little way from the center of the cornea. In another slide there was

shown an attempt at repair, and extensive vascularization was seen, polymorphonuclear cells giving place to a round-celled infiltration.

Another slide showed a similar condition, but less acute, in a guinea-pig, and here the state of affairs was only ascertainable by histological examination. Clinically, the cornea was intensely opaque throughout, and during life there was an irregularity in its center, but there was no clue to the nature of the condition until a section was made. This guinea-pig had probably been scratched by another on the eye. A section towards the edge of the wound showed an attempt at the process of repair, a downgrowth of epithelium filling up the edges of the wound. The whole of the rest of the cornea had largely lost its pattern, and there was a marked cellular infiltration. In the swollen part there was a dense network of capillaries, and a proliferation of connective tissue.

With regard to tuberculosis in the eyes of animals, he showed on the screen a section from a cat's eye. The animal was examined clinically before death, and one retina was found to be detached, and both eyes were blind. There was present a typical parenchymatous keratitis, and an intense infiltration with lymphocytes, with a mass of cells at the back of the iris. Cells were found scattered on the posterior surface of the cornea. Tuberculous parenchymatous keratitis occurred by extension from neighboring structures. Here was seen an almost staphylomatous condition at the corneoscleral junction.

The next slide showed the eye of a rabbit infected with tubercle from the subcutaneous inoculation of a tuberculous culture, of bovine variety. A tuberculoma was seen at the corneo-scleral junction. In another slide of the same eye there was marked papillitis, and the tuberculous process had extended into the optic nerve.

He also showed a drawing of a piece of the cornea of a dog which was affected with distemper. When the dog was seen clinically, the cornea was full of a network of vessels. In distemper there was a great variety in the eye lesions, as was the case too in tuberculosis.

Mr. HENRY GRAY, speaking as a veterinarian, gave a contribution on ulcerative affections of the cornea, their complications and sequelæ. He said that in the horse corneal

ulceration might occur in association with influenza, swine fever, trypanosomiasis, rabies, and from infections of the conjunctiva. In the dog this ulceration might be a sequel of paralysis of the optic division of the 5th, or of tumors involving the roots of the nerve. In the comparatively thick corneæ of the horse and cat, excavated ulcers were seen, but repair readily took place. These ulcers were accompanied by intense photophobia, lacrimation, and intense blepharospasm. For the last-named symptom he advised the instillation of cocaine drops with the object of relieving the conjunctival congestion.

A joint paper by Mr. A. F. MacCallan and Mr. Herbert Mason was read by the former, on cataract in horses, with references to iritis. It set out many interesting experiences of the authors in Egypt, where they had the advantages of an ophthalmic hospital and a Government veterinary school with an out-patient clinic, and a slaughter-house for most of the worn-out horses. Mr. MacCallan said that the mere presence of one or two opaque dots in a horse's lens might not interfere with the animal's efficiency for riding or draft purposes, but an extension of the opacity would render it useless for the saddle. Such animals should not be used for breeding, as most cataracts in horses were hereditary. It was not many horses on which it was worth while to do a cataract extraction. He described the operation he did for this extraction on the horse. Horses had a condition of iridocyclitis known as "periodic ophthalmia." Animals so affected suffered from photophobia, inflamed conjunctivæ, and had a hazy cornea, but not corneal ulceration. When this was present it was impossible to keep the pupil dilated with atropine. In severe cases of the disease the pupil became blocked. His own belief was that the disease was not contagious, and he had not found it possible to reproduce it in a healthy horse by inoculating humors from a subject of the disease. He thought it was an ocular complication of some general disease. Post mortem, besides the appearances already referred to, there were opacities of the lens, and flocculent opacities in the vitreous.

Discussion: General Sir John Moore spoke of diseases in the eyes of horses, basing his remarks chiefly upon his experi-

ences in France during the War, when he was responsible for the veterinary records. During the Great War there were 36,690 cases of eye disease in horses in France, 19,813 being cured, 2,558 were destroyed, and 9,723 were cast and sold, the carcases being much sought after as food. The majority of these affected horses had specific or recurrent ophthalmia, called by some periodic ophthalmia. This was regarded as so serious that a conference between French and British veterinarians was held, at which the disease was discussed. It was agreed that it was a recurrent iritis; the "moon blindness" of old coaching days. The animals with the disease mostly looked fat and well, and at a horse show some of them took prizes! At one period there were upwards of 2,000 animals under treatment for the disease. Some considered it was contagious, but the majority did not share that view. The bacteriological examinations which were done did not reveal any specific causal organism, and it was regarded as equally prevalent in horses' stables and in outside standings. The water did not seem to have anything to do with it. Each case received an injection of 1*cc* lugol solution into the supra-orbital fat, and the animals were subsequently evacuated to hospital. His own view was that the presence of much manure and urine had much to do with causation. 750,000 horses were taking part in the war, and 1.4% of the strength had this disease.

Major DUNKIN said that in dogs with distemper, eye lesions were common where the disease became chronic. In the ferret eye disease was one of the first indications of distemper. Keratitis in distemper in dogs was always preceded by a conjunctivitis, which was accompanied by a profuse secretion of tears.

Mr. M. B. OLIVER said he saw many cases of periodic ophthalmia in horses in France during 1918, and many of those animals were obviously ill; they had irido-cyclitis, with enormous exudates in the anterior chamber. He objected to the name, as it did not describe the disease.

Mr. HENRY GRAY, in reply, said that opalescent keratitis presented the appearance of opalescent glass, there was no vascularity, and it usually arose from distemper. Specific ophthalmia occurred during the Napoleonic wars.

Mr. GRAY CLEGG spoke of cases of hairy mole on the cornea in dogs.

Mr. HARRISON BUTLER referred to difficulties he encountered when doing a cataract extraction on a King Charles spaniel belonging to a friend. He said that on two occasions he had removed dermoid cysts from dogs' eyes, under cocaine, and there was no trouble.

Dr. RAYNER BATTEN also gave some experiences with eye diseases in animals, and remarked on the readiness with which interstitial keratitis was set up in the dog. Owners of dogs seemed very averse to anything operative unless an absolute cure could be promised.

Prof. WOOLRIDGE said that, in his hands, the treatment of glaucoma in the dog had been most unsatisfactory. He agreed that extensive opacity occurred in these animals on slight provocation. It seemed best to flood the eye at frequent intervals. Dermoids on the eyes of dogs were easy to treat; the growth could readily be dissected away.

Mr. E. TREACHER COLLINS spoke of a covey of pheasants at a shoot which refused to rise for the guns, and it was found that they could not see. One was sent to him for examination and he found it had a vascular cornea, there was typical interstitial keratitis and a large cellular exudation, with a marked development of vessels in the substance of the cornea. He asked whether veterinarians often saw sympathetic ophthalmitis in animals. He thought the horse disease which had been mentioned was better called "periodic ophthalmitis." He also had seen dermoid growths on the eyes of animals, and he had sections of a pig's eye with bristles sprouting from the cornea. In the Museum of the Royal College of Surgeons there was a specimen of a sheep's eye containing a tuft of wool.

Mr. TUDOR THOMAS spoke of a case of sympathetic ophthalmitis in a pet dog, one eye having received an injury, and the other eye was involved sympathetically.

Mr. A. L. WHITEHEAD questioned the reality of the supposed recovery of the clarity of the cornea in animals which seemed to have recovered.

Dr. G. MACKAY said he had been impressed with the facility with which keratitis occurred in the dog, but it also cleared up quickly. He showed a drawing of the case of a

fox terrier whose two lenses had come too far forward, and were spontaneously dislocated into the anterior chamber. In one eye the anterior chamber was quite filled with a dislocated and cataractous lens, and he undertook to remove the eye in which no pupil could be seen; but he miscalculated the size of the needed incision. Compared with the human, the dog's lens seemed hard, large, and round. No sepsis occurred.

An ordinary meeting of the Section took place on Friday, June 12th, the President of the Section, Sir ARNOLD LAWSON, K.B.E., occupying the chair.

CLINICAL CASES.

Massive Exudate.

Dr. RAYNER D. BATTEN showed a man aged 64 who had a massive exudate in the right retina, and some exudative changes in the left. He had been under observation since 1922, when he had an extensive vitreous hemorrhage in the right eye, and some retinal hemorrhages and white patches in the macular area in the left. The blood pressure was reported to be 135 to 85, there was neither sugar nor albumen in the urine. The central nervous system was normal, and the Wassermann negative. A year later the vitreous hemorrhage had cleared up, and the condition was as now seen, except that it had been very slowly increasing.

Cerebral Tumor Compressing the Optic Tracts, Chiasma and Nerves.

Mr. LINDSAY REA showed specimens and sections from a patient who had a tumor 40*mm* in diameter, yet who only presented sudden blindness of the right eye about Christmas time. A fortnight after the blindness he went to his family doctor and reported it, and Mr. Rea was subsequently asked to see him. He and eight others examined the eye, and there was nothing abnormal to be detected in the optic disc, and no change in the appearance or caliber of the vessels. Nasal

and neurological examinations yielded nothing, but a fortnight ago in the left eye—which had remained normal,—there was the beginning of papillœdema. As the pressure in his left eye was rising rapidly, he was X-rayed, and all that was seen abnormal was a flattened sella Turcica, and the erosion of one of the sphenoidal sinuses through the bone. Two days later the man died of a spreading meningitis, which spread from the base over the general brain substance.

Dr. W. E. CARNEGIE DICKSON, who made the drawing and did the post-mortem examination, said he thought the tumor probably originated in the pituitary body, but the specimen had not yet been cut and fixed.

Dr. A. FEILING said he saw the man at Mr. Rea's request and could find no cause for the blindness; there were no symptoms suggesting an intracranial lesion, nor of endocrine disturbance.

Mr. F. JULER remarked on the long time which was taken in the optic atrophy developing.

Mr. HERBERT FISHER discussed the case at some length, remarking that there might not have been any gross erosion of the sella Turcica, but some unusual anatomical arrangement of the cavernous sinus on that side, which enabled the growth to extend in that direction without much opposition. There had been gross stretching of the right optic nerve, and that had probably induced the early blindness, rather than a compression of the fibers of that nerve between the tumor and any bony structure at the base of the skull.

Mr. REA replied that originally the optic nerves were in their due anatomical relationship, but the sella Turcica was eroded and flat. There had been no disturbance of the visual field in the left eye, and there had been no approach to hemianopia.

Pulsating Exophthalmos.

Mr. A. E. A. LOOSELY presented a patient with a pulsating exophthalmos. When he first saw the case there had been diplopia and weakness of the external and inferior recti for 6 months. In each eye the vision, with correction, was $\frac{6}{9}$. When he saw the patient again a fortnight later there was pro-

trusion of the eye, the lids were brawny, and the eye could not be seen between them. A bruit was not only very obvious to the patient, but it could be heard by others also. Three days later Mr. Kerr tied the external and internal carotid arteries. There was no history of injury nor of venereal disease, Wassermann was negative, and X-rays showed nothing abnormal in the skull.

Epibulbar Sarcoma.

Mr. GEOFFREY VINER showed a lady, aged 31, who complained of a black spot in the left eye, which had been growing for 9 months. His diagnosis was based chiefly on his slit-lamp investigation. It showed pigmentation extending down into the deeper layers, chiefly the substantia propria.

Sir JOHN PARSONS thought this was probably a pigmented nævus or mole of the conjunctiva, a common situation for such tumors, which did not often show malignancy. Still, there were some unusual features, and he suggested that first the piece of conjunctiva should be dissected off, and have a microscopical examination made before deciding to operate radically.

Mr. ERNEST CLARKE agreed with Sir John. He had what seemed to be a similar case years ago, but a little deeper, and he removed the eye. Death occurred 5 years later from sarcoma of the liver.

Mr. G. H. POOLEY, of Sheffield, read a paper on **operative treatment of the lacrymal sac.**

He said those who had to deal with ocular troubles in a large industrial area had always many cases of dacryocystitis to deal with, and it was a condition about four times as frequent in women as in men. His aim was to find a quick and efficient method of dealing with these cases in a radical manner.

He classified the defects in this region which the surgeon might be called upon to treat, as follows:

Stenosis of the puncta,

Stenosis of the canaliculi just before they joined the sac,

Obstruction in the sac itself without retention or regurgitation of inflammatory products,

Obstruction with retention of inflammatory products which, on digital pressure, pass down the nose.

Obstruction with retention of inflammatory products which on pressure pass through the puncta into the conjunctival sac.

In all the foregoing, the inflammation appeared to be confined to the interior of the sac.

In a numerous and far more serious group the tissues round the sac were somewhat acutely inflamed, in many there was abscess and death of bone, many presented large red swellings, which might have burst before the ophthalmic surgeon saw the case.

The puncta might need incision or a three-snip operation Obstruction of the sac without regurgitation might be kept quiet without operation, but if necessary operation could be done.

Distention of the sac whose contents could be squeezed down into the nose required merely digital pressure by the patient, combined with the application of the antiseptic lotions.

Cases of distention of the sac the contents of which regurgitated on pressure but were apparently free from external inflammation gave good results if excised after having been injected with antiseptic dye. Even here a permanent drain into the middle meatus of the nose prevented epiphora.

Most trouble was caused by the suppurative cases. These he had been long content to drain, then foment until they were quiet, when he excised them, removing at the same time the lacryo-ethmoidal cells and any dead bone. In a good number abscesses subsequently formed, probably due to dead bone. And a disadvantage was the length of time required to complete the treatment, for it was irksome to the patient, and threw a heavy strain on a busy department at the hospital.

He next tried incision of the inflamed sac, removing the lacrymo-ethmoidal cells and scraping the nasal duct. This gave fairly good results, but there were some recurrences. Some of the patients were free from epiphora, but a larger number were troubled by it. There appeared to be no epiphora in the cases in which a free opening into the nose had been made when removing the cells or dead bone.

Late in 1923 he commenced making an opening through the sac into the middle meatus of the nose, scraping out the sac and packing firmly into the nose with ribbon gauze, this latter

being left in position three days. Two fishing-gut sutures which had been inserted but left unfastened at the time of the operation were now tied and the edges of the skin brought together. The operation only occupied three minutes, the results were satisfactory, and the convalescence rapid. Fluid was syringed through the lower punctum into the nose on the seventh and succeeding days until the patient returned home.

A further modification of the technique he had recently tried was, to make a curved incision half an inch long through the skin, beginning just below the tendo-oculi and going through into the sac. Then taking Lang's sharp spoon and dissector, he passed the dissector down the nasal duct then plunged the dissector into the cribiform part of the lacrymal bone and scraped out the lacrymo-ethmoidal cells. He then took a sharp gouge 4*mm* long on the cutting edge and cleared up the edges of the opening in the lacrymal bone, and extended the opening upwards and inwards to include the part of the nasal process of the maxilla which was behind the ridge. This opening he plugged with a bundle of thick thirty-day catgut; this kept open the aperture between the site of the sac and the middle meatus of the nose. He sutured the skin with two ophthalmic fishing-gut sutures, and if there were skin sinuses or dead bone, he removed them. In septic cases he fomented the wound afterwards, and syringed through the lower punctum on the 7th and subsequent days. He had now done the operation in 60 cases, some of them on both sides, and the results were most encouraging. There had been a few cases of recurrence of inflammatory trouble. In the end there was little deformity and little epiphora. The patients' ages varied between 18 months and 70 years. He had had two deaths: one from myocarditis during recovery from the anæsthetic, the other from a septic meningitis which had commenced before the operation was done.

Mr. W. B. OLIVER suggested that Mr. Pooley might try skin-grafting in place of the catgut plugs. MR. Gillie, for a similar operation, used dentist's wax, and covered that with a skin-graft.

Mr. A. D. GRIFFITH expressed his preference for the punch over the gouge for removal of the necessary portion of bone.

Mr. J. H. FISHER said he did not see much chance on the

operation described by Mr. Pooley having any effect on the epiphora, as there could be no permanent drainage through the puncta and canaliculi. The drainage which Mr. Pooley provided was of a temporary character, and was for the septic wound in which he was working. He did not think Mr. Oliver's suggestion as to a skin-graft was likely to constitute an improvement.

The PRESIDENT said he had hoped Mr. Pooley would have said more about epiphora, as this was not cured by removal of the sac. Most cases could be cured of the suppuration and the sepsis; it was the epiphora which gave the trouble, as in many cases this persisted as if nothing had been done.

Mr. E. D. DAVIS asked the experience of members in regard to the West operation for dacryocystitis. The successful cases following that operation which he had known were those which were acute. After Mr. Pooley's operation he thought there was danger of a fistula of the face from the nose occurring; he had seen one or two cases of the type in which a sequel of the operation was that mucus came on to the face from the nose.

Sir JOHN PARSONS said it did not seem quite clear whether Mr. Pooley operated on mucocele as well as lacrymal abscess. These were different conditions, and the same procedure was not applicable to both. The majority of mucoceles treated by excision of the sac, with clearing out of the upper part of the duct with a sharp spoon, did satisfactorily. As the excretory channels had been obliterated one could not expect the patients to be free of epiphora occasionally, especially in a strong wind, or in emotional stress. Intranasal operations for the condition seemed to be most unsatisfactory. The object of West's operation was to establish a communication between the interior of the sac and the nose. He had not experience of West's operation, but in most of the cases in which it had been done that he had seen the opening rapidly closed up, and one was no better off than before. Toti's operation seemed to have given better results. Mr. Pooley's operation was intended to cut short the treatment of lacrymal abscess, and it stood on a better foundation in regard to that condition. Where time allowed, he found no fault with the ordinary procedure of opening the abscess and packing it to the bottom,

and leaving it for a considerable time before doing anything further, even if there was a fistula. The pathology of lacrymal abscess was very difficult from that of mucocele. In the latter the trouble was confined to the interior of the sac, and in lacrymal abscess there was cellulitis, and if one interfered by operative measures without providing good drainage, the result was unsatisfactory. Mr. Pooley had introduced a method of securing good drainage into the nose, but the procedure seemed to be slightly risky. Unless one was overwhelmed with cases it seemed better to take a little more time, and, when the inflammation had subsided use the fistula as a guide to the source of the trouble, namely, the remnants of the sac which were left at the bottom. He doubted whether in many cases there was ethmoiditis associated with the lacrymal trouble.

Mr. AFFLECK GREEVES said he would have thought an opening into the nose was something to be avoided; he had seen two cases in which, following the making of such an opening, there was a general infection of ethmoid cells, and the patient had a chronic fistula, which did not clear up until the whole area was evacuated.

Mr. POOLEY, in his reply, said his use of dental wax for surgery in the war put him off using it since. Many of the patients he had to deal with did not come until the condition had developed an acute abscess, and in some the abscess had continued some time. He had given a good deal of thought to the question of the persistence of epiphora afterwards. He could not say how many years the opening he made would last, but he always syringed the cases as an after-treatment, as he wanted to make the opening durable. Probably the lacrymal gland did not secrete more tears than necessary, except in a strong wind and in dusty conditions. There were some borderline cases of mucocele which were about to become inflammatory externally, and in which an opening into the nose could be reasonably considered. He understood that the West operation, even in skilled and experienced hands, was a tedious one, and rather too much pneumonia had been said to follow it. If these cases were operated upon from the outside, through a small incision, there was no deformity, and the condition could be dealt with more thoroughly and easily

than through the nose. With regard to the formation of a fistula on the face, if one took away the edges of any sinuses which existed healing generally occurred; he had not had a single case of sinus as an after-result. He thought new operative procedures should be regarded from the point of view of the student, and the question asked "Is this better than what was done before? If so, how can we still further improve the procedure in the future?"

The Jullundur Operation for Cataract.

Dr. G. MONTAGU HARSTON read a short communication on the safety-limits of the Jullundur operation for the intracapsular extraction of cataract. He said that when Col. Smith introduced his operation for the intracapsular extraction of cataract, ophthalmic surgeons were much struck by the beauty of the operation, and the skill of the originator. That was his own case, and he at once proceeded to carry it out, reserving it for cases in native Indians and Chinese. He had some unsuccessful cases, and at first he could not understand the reason. Then it occurred to him it was probably a question of tension. Chinese were not nervous patients. He found that the safety condition for the operation was that in which the eye tension was the normal, i.e., between 18 and 25*mm* of mercury. Sometimes even 1*mm* tension in excess of normal sufficed to bring disaster to the operation as shown in the loss of vitreous. Since then he had done the intracapsular operation many times without fear of the results. He, the speaker, said he could do the operation far more easily with his fingers than with the Barraquer's apparatus; the former were more under one's control. The results of Col. Smith's operation, when successfully performed, were brilliant.

Sir JOHN PARSONS referred to his visit to St. Louis and watching Dr. Green operate by the Col. Smith method. He was not favorably impressed by the results; at the same time he thought "show operations" were a great mistake, as, under these conditions, it was difficult to fairly assess the value of a particular operation. His own view was that intracapsular methods must of necessity be accompanied by increased dangers to the patient's eye.

The PRESIDENT spoke of the need, in this operation, of a skilled and experienced assistant. He did not doubt that the combined operation was much the safer; it was the one he would choose for his own eye. After irrigation of the anterior chamber one could get as clear and black a pupil as by the intracapsular method. He would never do the Smith operation, certainly not on an European.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; M. J. Schoenberg, New York; T. H. Butler, London; P. G. Doyne, London; and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

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(*Continued from July issue.*)

III.—ANATOMY, PHYSIOLOGY AND PATHOLOGY.

56. Alexander, G. F. **Ocular sections.** *American Journal of Ophthalmology*, December, 1923.

57. Blatt. **Relations between intrauterine absorption of the opaque lens and microphthalmos.** *Zeitschr. f. Augenh.*, xlviii., p. 4.

58. Beauvieux. **Topographical and histological study of the zonule** *Archives d'ophtalmologie*, 1922, xxxix., Nos. 7 and 8.

59. Cirincione. **Development of the muscles and posterior layers of the iris.** *Annali di ottalmologia e clinica oculistica*, 1922, Nos. 1 and 2.

60. Duane, A. **The associated movements of the eyes, their nerve centers, conducting paths, production, varieties, and derangements.** *American Journal of Ophthalmology*, January, 1924.

61. Pinnoff, W. C. **Changes found in eyes of rabbits following injection of living tubercle bacilli into the common carotid artery.** *Ibid.*, February, 1924.

62. Fracassi. **A case of bilateral persistent pupillary membrane extensively developed.** *Periodico d'oculistica*, June, 1922.

63. Gillett, W. G. **The histological structure of the eyes of the soft-shelled turtle.** *American Journal of Ophthalmology*, December, 1923.

64. Gloor. **A case of congenital anomaly of the iris.** *Klin. Monatsbl. f. Augenheilkunde*, lxix., p. 126.

65. Hansen. **The occurrence of fat in the eye.** *Ibid.*, March, 1922.

66. Heine. **Ectodermal structures within the eye.** *Deutsche ophthalm. Ges.*, Jena, June, 1922.

67. Hoffmann, V. **Rapidity of diffusion of aqua ammonia in the eye.** *Archiv f. Augenheilkunde*, xci., p. 300.

68. Koby. **Horizontal asymmetries of the eyes.** *Revue Generale d'Ophtalmologie*, 1922, No. 2.

69. MERRILL, U. H. and WAGENER, H. P. **Anomalous spiral looping of the retinal vein.** *American Journal of Ophthalmology*, March, 1924.

70. PICHLER. **A rare malformation of the inner canthus.** *Klinische Monatsbl. f. Augenheilkunde*, lxviii., p. 378.

71. PIETRUSKY. **The behavior of the eyes in sleep.** *Ibid.*, March, 1922, p. 335.

72. PILLAT. **Persistent hyaloid artery.** *Zeitschrift f. Augenheilkunde*, xlviii., p. 43.

73. RIPPON, T. S. **The effect of tropical climate on physical and mental efficiency.** Royal Society of Medicine, War section.

74. ROEMER. **Connection of scrofulous keratoconjunctivitis with tuberculosis.** *Klin. Monatsbl. f. Augenheilkunde*, July, 1922, p. 128.

75. SEEFELDER. **Purulent conjunctivitis in a six months foetus.** *Deutsch. Naturf. u Aerzte*, Leipsic, 1922.

76. SEGUINI. **Comparison of the Sachs-Georgi and Wassermann reactions in the serum diagnosis of ocular syphilis.** *Annali di ottamologia e clinica oculistica*, 1922, Nos. 6–9.

77. STOEHR. **Innervation of the pial sheath of the optic nerve in man.** *Anat. Anzeiger*, 1922, Nos. 12 and 13.

78. TRIEBENSTEIN. **Malformation of the cornea and congenital anomalies.** *Klin. Monatsbl. f. Augenheilkunde*, lxviii., p. 578.

79. VAN DUYSE. **Apparent anophthalmos and colobomatous cyst pressing the upper lid forward.** *Archiv d'Ophtalmologie*, 1922, xxxix., No. 3.

80. WOLFRUM. **The structure of the anterior surface of the human iris.** *Archiv f. Ophthalmologie*, cix., p. 106.

ALEXANDER (56, **Ocular sections**) discusses the position and size of various corneal sections with especial reference to the available space, which is usually limited to the measurements of the inner part of the incision. ALLING.

BLATT (57, **Relations between intrauterine absorption of the opaque lens and microphthalmos**) bases his discussion on the following case. The left eye of a man 21 years old was perfectly normal, the right was about a third smaller than the left, otherwise well shaped. Remains of the pupillary membrane started from the lower outer part of the iris and ended free in the anterior chamber. Of the lens only traces of the membranous capsule could be found, which obstructed the pupil. Nothing of special moment was observed after dilatation of the pupil. The contents of the orbit were throughout smaller on the right side than on the left. The membranous remains of the lens formed a secondary cataract after the absorption of the lens, which Blatt thinks was brought about by a rupture of the capsule through a strong pull of the pupillary membrane

induced by some accidental trauma. The possibility of a spontaneous rupture of the capsule through swelling of the lens substance seems not to have occurred to him. The smallness of the globe and orbit was probably due to the loss of the lens. Vision was counting fingers at 1 meter. This poor vision also was probably to be ascribed to the loss of the lens, which caused a cessation of the formative and nutritive stimuli of the retina.

BEAUVIEUX (58, **Topographical and histological study of the zonule**) describes the observations he has made with the help of Zeiss' binocular loupe, Gullstrand's slit lamp, and microscopic sections. When a specially prepared anterior half of an eye is regarded from behind, the first thing to strike one is the complete independence of the lens and zonule from the vitreous. The zonule lies wholly in the posterior chamber, its free portion filling the perilenticular space. A fine transparent membrane covers the ciliary portion of the retina from the ora serrata and extends to the posterior surface of the lens. This membrane consists of a system of very minute fibers from the posterior part of the zonule and a transparent substance. If one looks at the eye from in front after the cornea and iris have been removed, he sees a delicate membrane running obliquely, instead of vertically like the preceding, composed of the same agglutinating substance and anterior fibers of the zonule. The anterior and posterior membranes unite at the base of the ciliary processes. A lateral view of the prepared eye reveals a triangular canal, the canal of Hanover. The canal of Petit lies behind the zonule. One sees the anterior and the posterior walls, composed of delicate membranes with amorphous basal substance. The lumen is filled with fluid distinct from the aqueous. If air is blown in the canal can be well observed with the loupe. The conditions are particularly distinct after staining the freshly enucleated eye with Mallory blue. The relations between the zonule and the ciliary processes, which are in direct contact, are likewise to be seen. For the microscopic studies eyes from infants and fœtuses were used. Meridional sections showed: 1. The anterior fibers of the zonule extend from their insertion in the equator of the lens in the form of minute bundles rather obliquely backwards, pass through the posterior third of the

ciliary despressions in their entire length and take their origin in the level portion of the pars ciliaris retinæ. Their thickness decreases toward the ora serrata and they have no connection with the crests of the ciliary processes. 2. The posterior fibers of the zonule are denser in construction and more complicated in their course. Part of them originate in the immediate neighborhood of the foot of the ciliary elevations, part accompany the anterior fibers to their origin at the orbiculus ciliaris. Isolated fibers extend like violin strings over the crests. A partial decussation of the anterior and posterior fibers takes place where the bundles come in contact with the base of the ciliary processes. 3. The equatorial fibers extend from the equator of the lens to the level portion of the pars ciliaris retinæ. All fibers which reach thus far originate in the ciliary epithelium and never extend beyond the ora serrata. The changed structure of the retinal cells in this region is peculiar. The fibers of the zonule are connected in no way with the hyaloid. Confirmation of the described conditions is obtained from other sections. In a section through the anterior portion of the ciliary process one sees no insertions of fibers, in one through the middle portion some insertions into the ciliary depressions may be seen, while sections still farther back show bundles of fibers running along the processes, and those through the region of the orbiculus ciliaris present evidence that here is where the fibers of the zonule originate.

With reference to the insertion of the fibers of the zonule in the lens, the delicate bundles of the anterior and posterior fibers come in contact with the cuticula superficialis of the lens capsule at a very acute angle, without influencing the form of the outer parts of the lens, and are inserted in a slightly wavy line. The pre-equatorial and equatorial fibers cause, by their tension at the place of insertion, a conical forward bulging of the outer lamella of the capsule in which the fibers end. The cone is filled with a substance supplied by the deep lamella of the capsule. At each point of insertion a fibril lifts up the limitans externa of the capsule, so as to give the equator of the lens a toothed appearance. In a 7 months old fœtus Beauvieux was able to demonstrate the two lamellæ. The origin of the zonule fibers lay in the orbiculus ciliaris; the accommodation fibers came from the level portion of the pars ciliaris re-

tinæ and the immediately neighboring depressions of the ciliary processes. The fibers which are inserted in the bottoms of these depressions and laterally in the ciliary processes are association and basal fibers. Beauvieux is of the opinion that fibers of the zonule originate from retinal epithelial cells. Many points of both physiological and clinical interest are omitted from this brief review.

CIRINCIONE (59, **Development of the muscles and posterior layers of the iris**) presents the following conclusions. The first trace of iris appears in the beginning of the fourth month in a separation of the free margin of the optic vesicle from the posterior corneal wall. This separation gives rise to a space filled with mesenchyma, which is divided into two portions, from which the great arterial circle of the iris develops. One portion of this gives rise to the stroma of the iris with Wachendorff's pupillary membrane. A fold of the optic vesicle is the starting point of the ciliary body. The free end to the anterior opening of the vesicle is the primordial iris, formed from the anterior pigment layer, which passes over into the posterior retinal layer at the free margin. At this place develops a zona germinativa for the longitudinal growth of both layers. It is therefore incorrect to designate the posterior epithelium as the pars iridica retinæ, as the retinal portion of the vesicle appertains to the ciliary body and is never pigmented, while the posterior epithelium of the developed iris is a product of the germinative zone. From this develops in the fourth month an epithelial plate which is in cellular union with both the anterior and the posterior epithelial layer. From this comes a muscle, the sphincter, through longitudinal growth of the cells and loss of pigment, which extends like a band between the epithelium and the stroma and in the course of the next month has its peripheral portion pushed off from its epithelial substratum by mesenchyma; yet the muscle remains in cellular connection with the zone germinativa by means of a narrow bridge until the end of the ninth month. These two facts the peripheral separation from the epithelium and the loss of pigment, have led earlier observers to overlook the epithelial origin of the muscle, in the opinion of the author. The preepithelial membrane appears first in the seventh month. In the sphincter free zone the anterior epithelium loses its cell

margin, the superficial cells lose their pigment, and send obliquely into the iris stroma protoplasmic processes which blend and form a homogeneous, non-nucleated membrana limitans 2 micra broad. This is constant and extends from the ciliary body to the sphincter. It can be sharply distinguished from the stroma, but is connected with the subjacent epithelium, from which it takes its origin. Frequently the blending of the protoplasmic processes is incomplete, and this has caused such modified processes with the epithelial cells appertaining to be mistaken for muscle fibers. Finally he says that the pre-epithelial membrane is the product of more or less advanced differentiation of the anterior epithelium. At any rate there is no muscle there with the functions which have been ascribed to a dilator, but a contractile epithelial membrane of muscle-like nature.

WOLFRUM (80, **The structure of the anterior surface of the human iris**) found places in which single non-pigmented connective tissue cells lay together, other places occupied by chromatophores, and wide areas formed only of collagenous fibrils. Increase of pigmentation he ascribes not to an increase of pigment cells in the iris stroma, but to an increase of lumps of pigment within the cells present. From the conditions he found Wolfrum believes that the anterior layer of the iris in man is in a state of retrogressive development.

Our knowledge of the nerve pathway concerned in ocular movements is far from complete but DUANE (60, **The associated movements of the eyes, their nerve centers, conducting paths, production, varieties and derangements**) presents what seems to be the most probable conception. Associate movements compose the conjugate in which both eyes follow at an equal rate an object moving in the frontal plane, or more strictly in the horopter, and disjunctive movements—convergence and divergence. For this classification, however, he prefers the terms unicentral and bicentral. The association centers in the cortex are: 1. The frontal probably at the base of the second frontal gyrus. 2. The occipital lobe close to the visual centers. 3. Temporoparietal centers located in the supramarginal superior temporal and angular gyri. The centers in the frontal lobe are for willed movements and are near the centers for corresponding movements of the head.

The occipital centers respond to the visual stimuli transmitted through the optic tract. The temporo-parietal are but transmitting stations. These centers not only discharge impulses causing muscular action but they also inhibit. The great avenue of connection between the various centers and the nerve nuclei is the posterior longitudinal bundle which decussates. Association pathways also connect the cerebellum and labyrinth. The author discusses associated paralyses showing that these may be divided into unilateral and bilateral. The former a paralysis of lateral movements, the latter of up and down movements, convergence and divergence.

ALLING.

According to FINNOFF (61, **Changes found in eyes of rabbits following injection of living tubercle bacilli into the common carotid artery**) lesions, as a rule occurring on the same side as the injection, appear after an incubation period of a little over six days. His experiments demonstrate that tuberculosis in rabbits of hematogenous origin may attack all the structures of the eye except the lens. The ocular lesions are of a severe and progressive type but with a few exceptions do not correspond to the form which are found in the human being. His results emphasize the well-known clinical fact that tuberculosis of the eye which occurs in subjects who have an active tuberculosis elsewhere in the body, is usually severe and progressive whereas the chronic types attack persons who have either a mild or latent tuberculosis.

ALLING.

GILLETT'S (63, **The histologic structure of the eyes of the soft-shelled turtle**) paper is a careful study of the turtle's eye illustrated by numerous drawings of microscopic sections.

FRACASSI (62, **Bilateral persistent pupillary membrane extensively developed**) describes thus the conditions found in a 15 year old girl. The pupil of the right eye was almost completely covered by the membrane, which was inserted by numerous trabeculæ on all sides in the little circle of the iris, and at the nasal margin became continuous with the stroma. The central portion was thin and opalescent, the margins thickened. Only a narrow crescent of the pupil was visible through the trabeculæ in the upper temporal part. Under

mydriasis the membrane became stretched so that a diffuse red reflex could be obtained through the central thinned part. Vision $\frac{1}{10}$. Almost exactly the same condition was present in the left eye, with an addition microphthalmos and horizontal nystagmus. Vision $\frac{1}{10}$. The membrane on each side was removed by operation after iridectomy, by which it was proved to be nowhere adherent to the lens capsule. Old choroiditic patches were now found in the fundus. The sight of the right eye became 0.4. The membrane did not essentially differ histologically from the attached bit of iris, except that the tissue had a more compact structure. The majority of the fairly numerous vessels were obliterated, the surface was rich in pigment cells.

GLOOR (64, **Congenital anomaly of the iris**) reports a case which has been under his observation from the 7th to the 28th year. The right pupil was displaced upwards, the left to the nasal side, both showed a weak but distinct reaction. The mesodermal layer of the iris alone was absent in places, which in other places there was complete absence of both layers causing polycoria through which the fundus could be seen. Glaucoms (hydrophthalmos) without enlargement of the cornea occurred in the 15th year and impaired the vision considerably. Cataract appeared in the right eye in the 28th year.

TRIEBENSTEIN (78, **Malformation of the cornea and congenital anomalies**) describes the following anomalies observed in a girl 12 years old. The right cornea was egg-shaped with the apex downward, indistinctly differentiated from the sclera by an opaque, vascularized zone. The iris was distinctly split into two layers, the anterior presenting an incomplete coloboma while the coloboma of the posterior layer apparently reached to the root of the iris. Typical coloboma of the choroid. Inverse course of the vessels and a crescent below the papilla. Absence of the fovea and of the foveal reflex. Great corneal astigmatism. Left cornea likewise deformed, pointing outward and downward. Iris divided into two layers, as in the right. The tissue of the anterior layer was much rarefied downward and inward so as to form a trough-shaped depression. At this place the posterior layer also was rarefied, the sphincter was absent, and the retinal pigment wanting from the pupillary margin to the ciliary body for a

breadth of about $\frac{3}{4}mm$. Coloboma of the choroid and of the optic nerve sheath.

HANSEN (65, **The occurrence of fat in the eye**) finds fat physiologically in the sclera, the lamina vitrea, the choroid, the ciliary body, and Descemet's membrane. He finds it pathologically in the arcus senilis, in the choroid in vascular diseases, sepsis, lipæmia, and miliary tuberculosis, in the retina in choked disk and nephritic retinitis. In the retina the fat lies partly inside and partly outside the cells.

HEINE (66, **Ectodermal structures within the eye**) describes three cases of adenoma. The first was an epithelial adenoma of the cornea in the angle of the anterior chamber following a minute perforating wound in a child 1 year old. Second, a ciliary epithelial adenoma in a case of microphthalmos with retinal cysts. Third, a pigment epithelial adenoma of the posterior segment in an eye with the sequelæ of endophthalmia after measles. He also reported two cases of apparent connective tissue growth from epithelia, one from the lenticular epithelium in a traumatic cataract, in which the newly formed tissue could not be differentiated from the corneal stroma; the other from the pigment epithelium of the fundus in a case of detachment, in which folds of pigment epithelium had apparently connective tissue contents.

KOBY (68, **Horizontal asymmetries of the eyes**) reviews the causes of asymmetry of the human body and finds many factors taking part, but two of particular importance. These are heredity and the more or less intensive use of the organ. Intrauterine deformation of the skull by the pressure of the pelvic bones also takes part in the production of asymmetries of the head. Heredity plays the great part in asymmetries of the face. On the basis of two cases Koby distinguishes two main types of horizontal asymmetries of the eyes; an asymmetry of the bones, which may be of fœtal origin, and an anomaly of the soft parts, which is due to heredity in certain cases.

PICHLER (70, **Rare malformation of the inner canthus**) found in a man 69 years old a fistula-like opening to the nasal side of the caruncle through which a probe could be passed into an apparently closed pouch as large as the head of a pin. No connection with the lacrimal passages could be traced.

PILLAT (72, **Persistent hyaloid artery**) reports the case of a man 24 years old who had several malformations in his right eye, among them a persistent hyaloid artery. The cornea was smaller, the anterior chamber shallower than the left. There were numerous opacities in the posterior cortex. Convergent strabismus. The persistent artery came from the middle of the papilla, ran at first forward and upward, then bent down and in at an acute angle and suddenly broke up into several branches which ran to the posterior surface of the lens. Colobomatous changes in the choroid were present above and below the papilla. A notable feature of this case was the fact that in spite of the many malformations, and although the field of vision was concentrically contracted, the eye could still fix.

VAN DUYSE (79, **Apparent anophthalmos and colobomatous cyst pressing the upper lid forward**) found in a child 5 weeks old the upper lid pushed forward by a soft, fluctuating tumor, situated between the lid and the palpebral conjunctiva. No trace of a microphthalmos was perceived. The cyst had grown rapidly for three weeks. Before excision 6*ccm* of yellow fluid was obtained by puncture, which chemically resembled a transudate. Recurrence necessitated a second removal three months later. The cyst removed at the first operation measured 2.3 × 2 × 1.5*cm* and showed microscopically no traces of an eyeball. The cavity of the cyst was enclosed by two layers. Next the cavity was a much folded glial layer, thought to be a duplicature of the embryonal retina, then a layer of glia and connective tissue, vascularized and containing muscle tissue. The proximal part of the tumor was formed of cells having a marked blastomatous character. At the second operation the tumor was as large as an olive, not cystic and composed of more or less vascular neuroglia tissue, the nuclei being of many forms. No cornea, lens, uvea or optic nerve was present, the sclera only in places. The retina was incomplete, the external granular layer only with a rudimentary internal, and no nerve fibres, ganglion cells, or rods and cones.

The case reported by MERRILL and WAGENER (69, **Anomalous spiral looping of a retinal vein**) showed the superior nasal vein starting normally in the periphery but when a short distance from the disk turning sharply into the vitreous forming

a loop about 2.5*mm* in length. On its return to the retina it passed into the central vein. A strand of glistening white tissue was observed accompanying the vessel. About 30 cases of this sort are on record. It is thought that these anomalies are examples of extreme tortuosity of the vessels and that they have no relation to the hyaloid artery.

ALLING.

STOEHR (77, **Innervation of the pial sheath of the optic nerve**) finds two sorts of nerves, those to the pial vessels coming from the carotid plexus, and those to the pial connective tissue, twigs from the oculomotor nerve, which are by far the more numerous. The nerve fibers of the pial connective tissue have no end formations, but divide into loops and meshes, in the formation of which neighboring fibers are involved, so that they blend in the periphery and form a closed network.

ROEMER (74, **Connection of scrofulous keratoconjunctivitis with tuberculosis**) concludes from a study of fifty cases that scrofulosis of the eye is always associated with demonstrable tuberculosis of the lungs.

SEEFELDER (75, **Purulent conjunctivitis in a six months' fœtus**) found a purulent conjunctivitis of the right eye of a six months' fœtus in spite of the fact that microscopically the lids were still closed and appeared to be completely grown together. The microscopical examination revealed minute apertures in the luting together of the lids, through which the agent must have entered. Bacterium coli was found in the pus.

HOFFMANN (67, **Rapidity of diffusion of aqua ammonia in the eye**) observed a case in which an 8.8% solution of ammonia was acidentally thrown into a patient's eye. At first there was little change even in the eye which suffered most. The cornea was not opaque, but dull from epithelial defects, the pupil contracted from irritation of the iris, the lens clear. During the following days a severe iritis developed with hypopyon, the cornea began to grow opaque, the lens swelled rapidly until it nearly obliterated the anterior chamber and became opaque, and the tension rose to 55*mm* of mercury. In spite of an iridectomy and removal of the lens substance a complete anterior synechia and cicatrization of the cornea took place. The malignant and insidious course corresponds with that de-

scribed by other authors, and Hoffmann tried to learn experimentally why the serious consequences of burns with ammonia set in so late. These experiments showed that ammonia very quickly penetrates into the inner parts of the eye without primarily causing serious symptoms on the part of the cornea, iris, or lens, so he assumes that it induces serious metabolic injuries which in turn give rise to the gross intraocular changes.

SEGUINI (76, **Comparison of the Sachs-Georgi and Wassermann reactions in the serum diagnosis of ocular syphilis**) made these tests on 198 cases. Both tests gave positive results in 38, negative in 147, while in seven the Wassermann was positive and the Sachs-Georgi negative, although the patients were suffering markedly syphilitic diseases. In six cases the Sachs-Georgi reaction was doubtful. The two reactions agreed in 93% of the cases.

PIETRUSKY (71, **Behavior of the eyes in sleep**) made the following observations on 300 sleeping men of various ages. The position of the eyes in sleep varies a great deal, but most commonly they are turned up and diverge. In deep sleep eye movements may not rarely be observed; these are often not uniform in the two eyes, and when they are associated they are often unequal. At the moment of awakening the eyeballs suddenly take the middle position with a sudden dilatation of the pupils. The eyes then never fail to turn away from light. These movements occur in both adults and children. The deeper the sleep, the smaller the pupils, yet the light reaction can be obtained even in very deep sleep. Dilatation to sensory irritation is prompt, especially in light sleep. With convergent movements of the eyes a contraction of the pupils in sleep is not perceptible. At the moment of awakening the pupils dilate strongly even though intensely illuminated. In infants the pupils are not as small during deep sleep as those of adults, and dilate less markedly. Reaction to light and sensory irritation are present a few days after birth.

During the course of a year's experience with the R.A.F. in India RIPPON (73, **The effects of tropical climate on physical and mental efficiency**) noticed that there was a tendency for pilots, who in Europe were quite efficient, to make bumpy landings. On examination he found that these men showed an inability to maintain an even ocular muscle balance. This

discovery stimulated further investigation. He made particular examination of all cases who complained of discomfort due to glare. An inspection of the symptoms of which the patients complained showed that they could roughly be classified into three groups:

1. Superficial irritative group.
2. Retinal fatigue group. (Night-blindness, prolonged after-images and slow adaptation.)
3. Cases unable to sustain efforts of accommodation and convergence for near objects for any length of time. (Accommodative asthenopia.)

With regard to accommodative asthenopia his investigations indicate that the first sign of visual fatigue is shown by fatigue of accommodation and that though it is only in cases of marked accommodative asthenopia that faulty landings occur yet this sign is an important sentinel of the early onset of eye fatigue. With regard to the effect of glare on convergence it was found that weakness of convergence was directly correlated with length of service in tropical conditions.

Rippon points out that eye fatigue which in a beginner would be sufficient to cause faulty landings, in an experienced pilot may be compatible with safe flying. Nevertheless, the early recognition of the onset of ocular fatigue and the timely ordering of rest is of the greatest importance in preventing the onset of "anxiety neurosis" where the predisposing cause is the eye fatigue and the constant fear of crashing on landing.

P. G. DOYNE.

(*To be continued.*)